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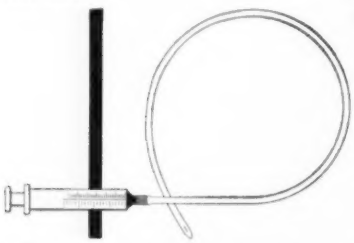

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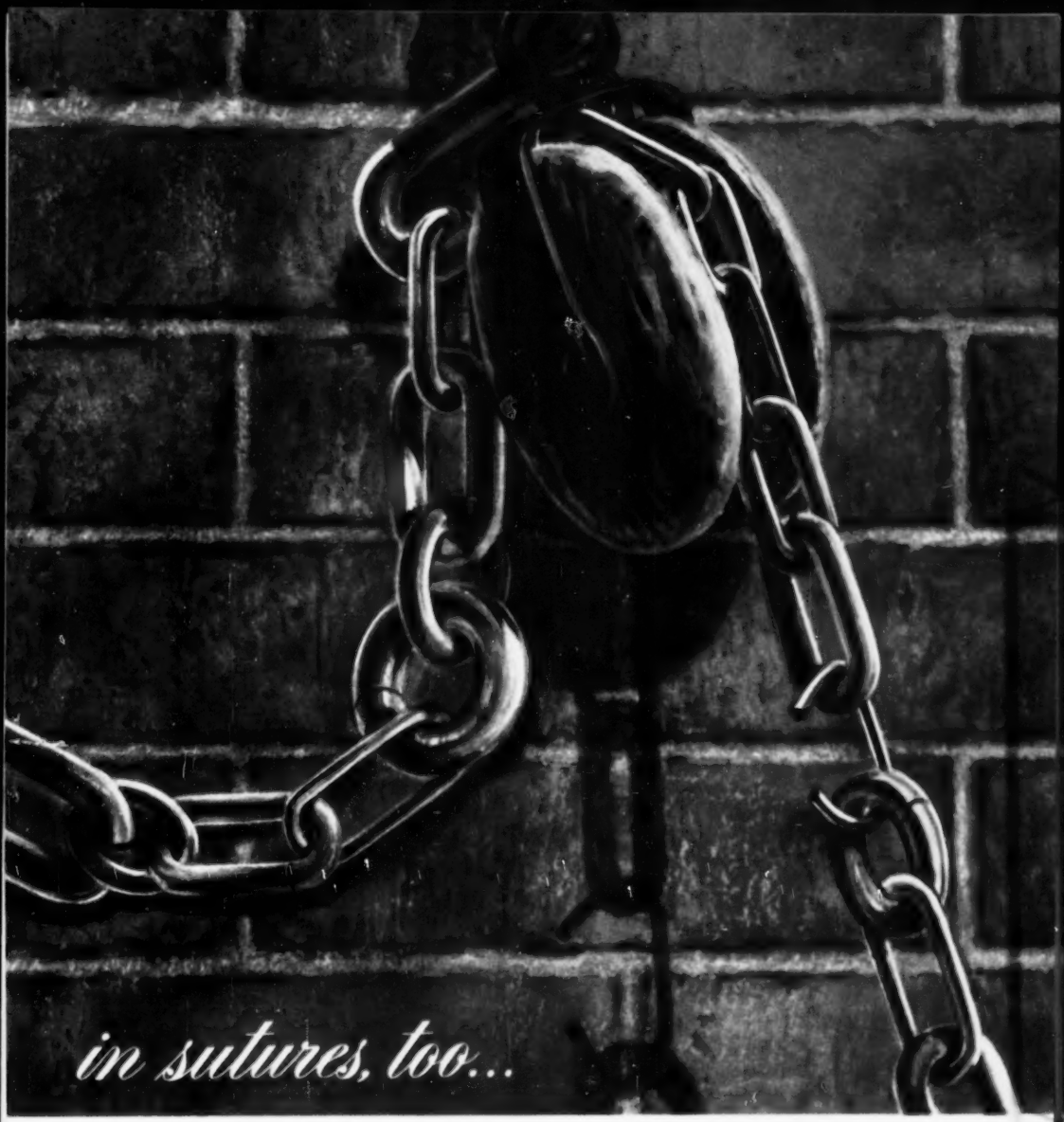

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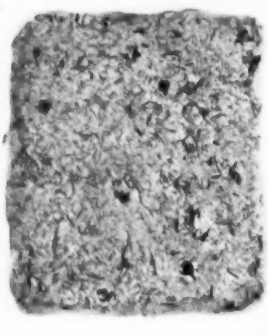

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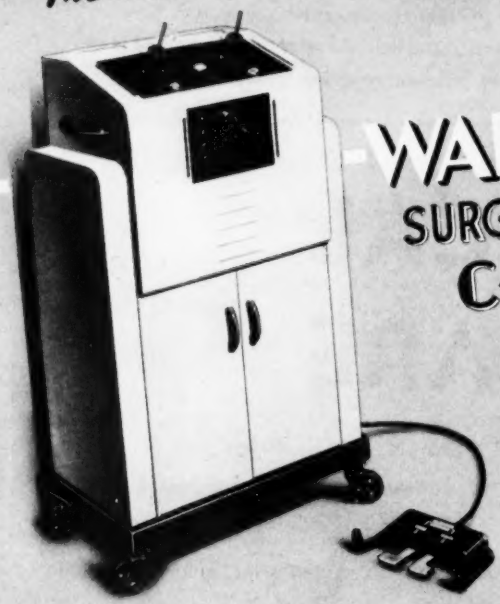

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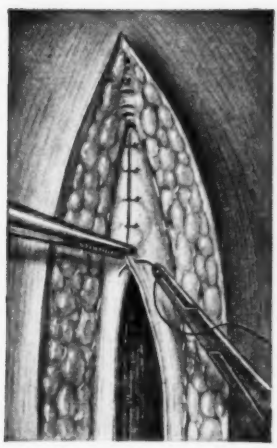

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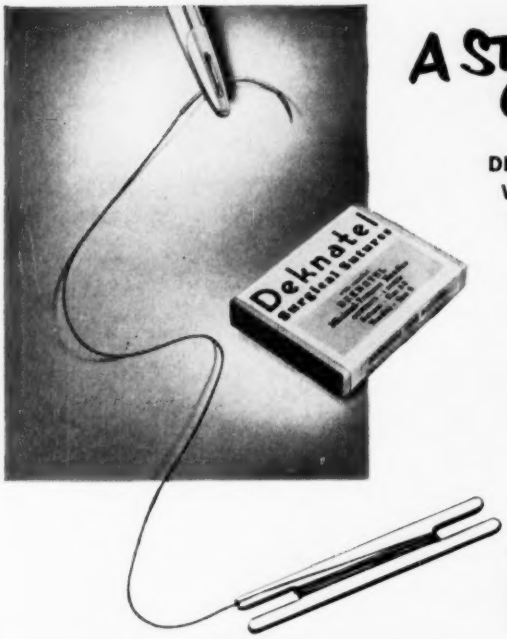

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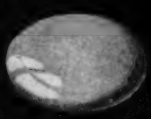

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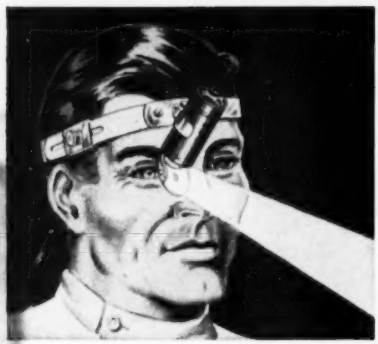

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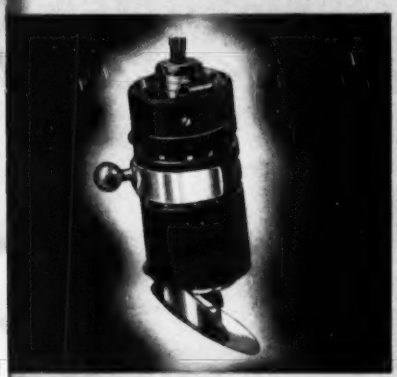

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VOLUME 65 SEPTEMBER 1952 NUMBER 3

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Editorials

INTRAVENOUS ANGIOGRAPHY

IN SPITE of care and experience, we still find the making of aortograms a rather major undertaking. We have reserved it for the urologic diagnostic problem which cannot be solved by other means.

Intravenous angiography, using 50 cc. of 70% sodium acetrizoate (urokon® sodium) injected in the cephalic vein in two seconds by means of a simple home-made lever can help fill the gap between the usual IV urography or retrograde pyelography and the more complicated aortography. We have demonstrated to ourselves that if an intravenous drip of 0.6% procaine hydrochloride is started before injection, the peripheral flush and subsequent headache and nausea are suppressed. Timing of exposures has been the most difficult problem, for a one-second delay may find the column of dye already in the iliac vessels, whereas exposing the film too soon demonstrates only the chambers of the heart. Serial films with a rapid cassette changer are helpful. Ten seconds is most often the correct interval from the end of the injection to the arrival of the dye in the renal vessels.

However, even if the angiogram has been missed, exposure of a film 25 seconds after injection will make the kidney opaque in an intense nephrogram. The dye then has been taken up by the cells of the renal tubules, and space-consuming lesions may be outlined. Exposure of films at one and five minutes will give excellent urograms because of the high concentration of dye which has been administered.

We don't know yet the place of this relatively simple technique in the work-up of the patient, but it may be used more frequently in the diagnostic problem for which ordinary intravenous urography is inadequate and as a valuable supplement to retrograde pyelography.

FRANK HINMAN JR., M.D.

From the University of California School of Medicine.

SURGERY IN THE OHIO VALLEY

Its Importance in the Development of American Surgery

B. NOLAND CARTER, M.D.
CINCINNATI

ALTHOUGH surgery as we know it today was impossible before the widespread employment of anesthesia and aseptic principles, its complex history extends well into antiquity. American surgery, however, can be said to date only from 1768, at the very earliest, when the first class was graduated from the Medical School of the University of Pennsylvania, the first American school of medicine to be established. Even the training of these men was carried on by European professors, according to European concepts, and was dependent on European instruments and supplies. Independent development was possible only after the American and French Revolutions had disrupted connections between the Old and the New Worlds. In this development, Britain and France were the principal sources of influence. Finally, it was with the opening of the trans-Allegheny Territory to settlement and with the beginning of the westward pioneer movement that American medicine and surgery really began to develop along independent lines.

Since the chief avenue for settlement of this newly opened territory was the Ohio River, and since it is along its valley that many of the major developments took place, it is this geographical area with which we shall be chiefly concerned. Three stages of development in surgery may be noted: the pioneer stage, when the medical frontier was literally unlimited; the developmental stage, when villages had grown into towns and hospitals and medical schools were founded, and, finally, the scientific stage, when technical developments had laid the foundation for modern surgery and when the medical frontiers remaining for exploration began once again to expand. By the time the Civil War exerted its disruptive influence, the modern period had begun.

The Ohio Valley has been selected as a microcosm of the American surgical picture as a whole, for geographical, social, and political reasons. It was the first avenue for settlement of the new territory, isolated from Europe and, in large measure, from the eastern seaboard which was in communication with Europe. This isolation tended to increase reliance on individual ability and judgment and to create what is often regarded as the typical American attitude and character.

The pioneers who pushed into the Ohio Valley and through it into the South and West were a hardy people, diversified in background and experience and cultural patterns. In consequence, no single medical group or school was able to dominate, and early practice was eclectic and independent. Regular physicians,

Presidential address, read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

"botanics," and quacks lived and worked side by side, judged by the layman solely on the basis of the results of their methods of treatment. Surgeons and physicians were thus emboldened to attack problems for themselves instead of accepting previous dicta without question.

The pioneer was largely responsible for his own medical care. Equipped only with a small store of drugs, he soon learned the local medicinal lore of herbs, roots, and barks and added this knowledge to what he had brought from the eastern seaboard. Significantly, the first medical book published in the West was Peter Smith's "The Indian Doctor's Dispensatory," a treatise on the gathering and use of native remedies. The first regular practitioners in the new territory were Revolutionary Army surgeons, who were attached to the militia sent to protect the pioneers from hostile Indians, or who had been drawn west by the liberal land grants with which many were rewarded. The practice of medicine as a settled profession followed not long after.

The crudity of conditions faced by the first physicians is almost unbelievable. Often a physician's complete equipment for both emergency and standard practice could easily be contained by the saddlebags on his horse. Distances to be covered were sometimes so great and roads, if any, were so poor that in one instance a physician reportedly spent two weeks making the circuit of his necessary calls before he was able to return for his second visit to a seriously ill patient. Under such conditions, heroic surgery was the rule rather than the exception. Isolated by great distances from one another, physicians were forced to depend on their own initiative, common sense, and the logical application of established principles to new situations.

If the frontiers were broad, the tools for combating disease and accident were few and crude. The principal remedies were bloodletting, purging, and poulticing. The latter two comprised a formidable battery of drugs and concoctions, including calomel and jalap for purging and tobacco and elm bark for poulticing. Standard surgical techniques, which were even fewer in number, consisted of lancing of abscesses or swellings, amputation of gangrenous or macerated limbs, and excision of fungating or ponderous tumors. A dislocated shoulder could be reduced "with the aid of four strong men." Because of the violence of his measures, both medicinal and surgical, as well as the frugality of pioneer life, the physician was seldom, if ever, called until the home remedies had been unsuccessfully exhausted.

As the pioneers settled into villages and towns, the phyiscian likewise became more settled. Adventurous and ambitious men continued to push westward, but the need for some method of educating local young men in the practice of medicine soon became apparent. The population of most of the new western towns was so diverse in origin and background that there was no single continuing influence to direct their progress or to impede it by sheer weight of tradition. The immediate answer to this situation was the so-called preceptor system. With this method, a young man was bound to a physician for a definite period of time, usually two to three years. During this period, as in any trade apprenticeship, the youth served as errand-boy and general assistant. If, as was usually the case, the physician prepared his own drugs and medicines, the pupil might be set to grinding quicksilver into mercurial ointment while reading, for example, Quincy's "Dispensatory." No doubt he found the latter the more difficult of the two tasks, as did young Daniel Drake in Cin-

cinnati in 1800. Practical experience thus emphasized what might have been learned from the few precious volumes in the preceptor's library, frequently meager indeed. Often the student accompanied the physician on his calls, sometimes to assist, and sometimes merely to hold the horse.

After he had absorbed the contents of volumes like those Drake remembered, such as "Chesselden on the bones and Innes on the muscles, without specimens of the former or plates of the latter," [1] the young apprentice might or might not be given books on chemistry, botany, or physiology. At any rate, there was no dissection from which to learn anatomy nor any scientific study underlying the etiology of disease. Empiricism was the order of the day, and the greatest boon a preceptor could confer on his pupil was to allow him to assist in the many treatments which he performed and to give him the chance to observe the effects of each. Thus, the wider a preceptor's practice, the more a lad might learn, always supposing, of course, that the physician was interested in his charge's professional education and not merely in having him as an inexpensive helper.

During the pioneer period, the medical frontier was virtually unlimited. The physician practiced when, where, and as he pleased, moving on from one place to another if he felt his freedom was threatened. He needed no state license; more often than not, he had no formal education; he needed only physical hardihood and a strong urge to aid his fellow man. Indeed, if one considers the existing fees and necessary expenditures of the day, it is immediately apparent that love of profession and of humanity were the prime motivating forces to these men whose hardships were many and whose leisure and worldly returns were little.

As means of communication improved, the communities became more closely knit, and medicine along with socioeconomic conditions entered into its second, or developmental, stage. Reports in the scientific periodicals of Europe and the eastern United States of discoveries and advances in medicine and surgery impelled the pioneer physician toward a closer scrutiny of his own performance and progress and toward the establishment of tentative standards.

In March, 1822, a quarter century after the publication of the first American scientific periodical, *The New York Medical Repository,* John Godman, of Cincinnati, launched the *Western Quarterly Reporter of Medical, Surgical, and Natural Science.* This was the first journal of its kind to be published west of the Alleghenies. The *Quarterly* lasted through six issues; three years later, in 1826, a second publication, *The Western Journal of the Medical and Physical Sciences,* was begun, also in Cincinnati, continuing there for thirteen years under several titles. It was thereafter moved to Louisville, with its then existing editor, Daniel Drake. Aptly, its motto was "E sylvis aeque atque ad sylvas nuncius" (the messenger from the woods as well as to the woods). The bulk of its articles were from local correspondents—truly "from the woods"—or were condensations of Eastern or foreign communications—"to the woods." Meanwhile, in 1828, the *Transylvania Journal of Medicine and the Associate Sciences* was begun in Lexington, Ky. By 1851, over a third of the thirty medical periodicals mentioned by Drake in his "Discourse Before the Library Association" were published in the trans-Allegheny Territory.

1. Drake, D.: Discourses Delivered by Appointment Before the Cincinnati Library Association, Cincinnati, Moore & Anderson, 1852, p. 56.

Although the limitations of the preceptor system became increasingly apparent to physicians of discernment, the journey to Philadelphia for regular courses of lectures at the University of Pennsylvania still remained a formidable obstacle to many young men. Dating from 1799, the Transylvania Seminary in Lexington had had a medical department with two professors, but it was not until the second decade of the nineteenth century that it assumed significance as a medical college, ranking second in 1826 on a list of fifteen medical schools. On the same list, Daniel Drake's venture in Cincinnati, the Medical College of Ohio, appears last, although a group of twenty-four young men had graduated from it in 1821, five years previously. At this time, Transylvania was the center of medical education in the West. Its greatness stemmed in large measure from the members of its faculty, principally, Benjamin Winslow Dudley, Professor of Surgery. At varying periods other notables, such as Daniel Drake and the botanist Rafinisque, also added to its stature. Its decline after Dudley's resignation was rapid, partly because of Lexington's location, which became disadvantageous as the river trade assumed increasing importance. Without the fame of Dudley as a drawing card and with a faculty divided on the question of moving the medical department to Louisville, the glory of Transylvania passed.

As for the Medical College of Ohio, torn by factional disputes, it was by turns brilliantly staffed and well attended or was marked by mediocrity in faculty and curriculum and attended by few students. It had, however, from the very beginning, the advantage of a hospital connection; it was in this regard the first such school west of the Alleghenies and, apparently, the second in the whole United States. In his foresight, Daniel Drake had secured from the Ohio Legislature not only funds for a medical library and equipment for the study of anatomy but also a charter for the Commercial Hospital and Lunatic Asylum of Cincinnati. One of the provisions of the law establishing this hospital, was that the faculty of the Medical College of Ohio must give free

> . . . medical and surgical advice and service which their cases may require, in consideration of which they shall be at liberty, under such regulations as they may choose to prescribe, to introduce the pupils of said college into the said hospital, to witness the treatment of patients there assembled.[2]

The record books of the hospital reveal that as late as 1847 the faculty deliberated in the diagnosis of certain cases and that on their recommendation operation was performed by the Professor of Surgery "in the presence of the students and faculty." The faculties of the Medical College of Ohio and the Miami Medical College, both of which Drake founded, comprised at one time or another, in addition to himself, such personages notable in early American surgery as Samuel Gross, Willard Parker, Reuben Mussey, and George C. Blackman.

The Ohio territory, at this time the crossroads of the nation and the point of convergence of different social cultures, soon also became the major battleground for conflict between regular medicine and the various cults then in existence. Most important among these fads was Thomsonianism, which attempted to reform medicine by using only "natural" remedies, principally herbal concoctions and steam baths. In 1830, this group took over the failing Worthington College near Columbus,

2. Section VIII of law enacted by the General Assembly of Ohio, Jan. 22, 1821, quoted in Bachmeyer, A. C.: The Hospitals of Cincinnati During the Last Century, Univ. Cincinnati Med. Bull. **1**:27, 1920.

which had never had a medical department. In 1832, an early cultist periodical, *The Thomsonian Recorder,* had been successful in engineering repeal of the Ohio Medical Practice Act. By 1835, nearly all the states had repealed or amended their practice acts in order to allow the "steam doctors," as they were derisively called by the regulars, the right to practice. Empiricism ran high; the frontier was still open, and the chief authority was that of the testimonial. In 1841, the Thomsonians moved to Cincinnati, where they called their school the Botanico-Medical College and later, the Eclectic Medical College, which continued to exist as late as 1939.

The eclectic, botanical, and other nonregular schools were able to flourish unchecked because of the general absence of laws regulating practice and the weakness of those that were in existence. In 1811, Ohio had been divided into five medical districts, each with three medical censors to examine candidates for certificate. Only certified physicians were able to apply for legal assistance in collecting bills. Although fines for illegal practice were fixed as early as 1812, they were virtually worthless because there was no legal machinery for their enforcement. When in 1832 the botanical-eclectic group secured repeal of even these inadequate measures, Ohio was left without any law regulating the practice of medicine. This situation continued until 1868, and it was not until 1893 in Kentucky, and 1900 in Ohio, that examinations by a state board of licensure were mandatory.

Prior to the establishment of legal requirements, prominent physicians in the individual towns had formed medical groups for the promulgation and improvement of medical education and for the elevation of professional standards. Because of inadequate transportation facilities, district meetings were more frequent and important than the state-wide conventions. Early topics under discussion included professional etiquette, recommendations to the legislature for a law requiring vaccination for admission to the public schools, means of obtaining an adequate supply of leeches, and petitions to the legislature for amendment of the criminal law to provide bodies of those convicted of capital crimes for anatomical dissection.

One of the major problems of the early medical colleges in America was the procurement of cadavers for dissection in anatomy courses. Such procurement was not legally possible, despite the recommendations of the 1835 Medical Convention of Ohio that the bodies of those convicted of capital offense be turned over to the medical colleges. Public feeling ran high on the subject; mob violence took place in New York City as early as 1788, and the pillaging of Worthington College in Columbus by an irate crowd caused the closing of that institution in 1839. It was not until the scandal of 1878 occurred, when a grandson of President William Henry Harrison walked into the dissecting room of the Ohio Medical College on an errand and saw the body of his own father, the President's son, on the dissecting table, that steps were taken to secure a legal supply of cadavers.

As may be surmised, there was in this period no scientific basis for medicine and surgery. Treatment in the crude hospitals of the time was largely empirical—either a remedy was effective and was continued, or it was not effective and was replaced by another. There was, prior to 1815, no hospital in either Cincinnati or Louisville, the two largest towns of the area. In 1815, a private house was rented in Cincinnati for the care of the sick who could not be tended at home. Two years later, in Louisville, a hospital was chartered for the care of boatmen who needed treatment while passing through that increasingly important river town. In 1823,

the Commercial Hospital and Lunatic Asylum opened its doors for the care of boatmen and commercial travelers taken ill while in or near Cincinnati. From the ward records of this institution, one may get a truer picture of the surgery of those times than from the written accounts of operations performed by daring, though ofttimes ill-equipped, surgeons.

The prowess of a few surgical giants, such as Valentine Mott, of New York, Philip Syng Physick, of Philadelphia, John C. Warren, of Boston, Ephraim McDowell and Benjamin Dudley, of Kentucky, and Marion Sims, of Carolina, is already familiar. Less well-known surgical pioneers like John Richmond, of Newtown, Ohio, also attained some fame, though chiefly local. A member of the second graduating class of the Medical College of Ohio, Richmond, it is related, was called out on a raw spring night, while flood waters were raging, to attend a young woman who had been in labor for an abnormally long time. Arriving at the woman's cabin, he found two midwives who could tell him only that "she had fits and the pains did no good." The night was so stormy that the women had to hold blankets up to keep the wind from extinguishing the candles, which were the sole means of illumination. Additional help could not be obtained because of the high water, nor could "cordials" be procured to mitigate the patient's suffering. As the only means of saving his patient's life, Richmond undertook a Caesarean section, delivering her of a dead child. The operation was successful, and the patient returned to hard work twenty-four days after the delivery. This was the first Caesarean section performed in the United States and the first one west of the Alleghenies.

On the other side of the ledger, Alexander Dunlap, of Springfield, Ohio, who performed an ovariotomy thirty-four years after McDowell, was denounced by the profession in 1843 as having undertaken an unsafe and unjustifiable operation. A review of the surgical ward records of the Cincinnati Commercial Hospital from 1839 to 1845 helps to explain this attitude. There was an overwhelming majority of admissions to the surgical wards during this period for such quasisurgical diseases as ulcer, abscess, venereal lesions, or, occasionally, wounds of violence. The only procedures frequently recorded are amputation, fistulectomy, hemorrhoidectomy, and various operations for the removal of cataracts. In the case book covering the period Aug. 24, 1843, to January, 1845, only fifteen surgical procedures other than incision for abscess are recorded. These are as follows: three fistulectomies, two excisions of cataract, and one each of hemorrhoidectomy, hydrocelectomy, herniorrhaphy, tracheotomy, removal of a wen, removal of an ingrown toenail, amputation of a finger, excision of a cancer of the lip, and reduction of a dislocated hip. Surgery in this period could hardly be termed an important specialty!

The surgical history of this developmental period is largely the individual history of such intrepid pioneers as Daniel Drake, Benjamin W. Dudley, Willard Parker, Ephraim McDowell, Alban Goldsmith, Samuel Gross, Reuben D. Mussey, and George C. Blackman. Hence a thumbnail sketch of their careers is indicated at this point.

Of this group of doctors, only Daniel Drake was not a surgeon, but his towering influence cannot be ignored. It was he who organized the medical faculties in Cincinnati which attracted such men as Parker, Gross, and Blackman, and, for a shorter time, Horatio Jameson. Drake secured the charter for the first hospital in Cincinnati, and all his life he was a leader in the fields of medical education,

medical periodicals, medical legislation, and medical organizations. He was the most truly scientific of the early group, publishing systematic accounts of his research into botany, topography, and endemic diseases. He investigated the caliber of medical education in his time and pointed out needed reforms and founded during his lifetime two medical colleges, serving as professor in three others. He was one of the first in America to assert the plausibility of minute organisms, or animalcules, as the cause of cholera. A civic-minded cultural leader, Drake did much to improve Cincinnati and to stimulate interest in it on the part of prospective settlers. He was active in amateur theatricals and in the organization of a literary debating society. His sense of the dramatic, as well as his innate dignity, is revealed in his own account of his expulsion from the faculty of the first medical school he founded. As president, he occupied the chair and put the vote regarding his expulsion to the three-member faculty meeting. His resignation was demanded by a 2 to 1 majority, he himself casting the negative vote In later life, he delightedly referred to the great dignity with which one of his two ex-colleagues immediately lighted him downstairs with a tallow candle as one of the most humorous incidents in his checkered career. Public sentiment demanded his reinstation, but his own pride forced him to resign.

In founding a rival institution to this first school, Drake attracted to the West such men as the famous Samuel Gross and Willard Parker, who was to be later the first surgeon to perform cystostomy for irritable bladder and the first American to lead in the proper care of appendicitis. In 1827, Drake established the Cincinnati Eye Infirmary and became known as a bold and skilful ophthalmic surgeon, but his chief fame and his greatest satisfaction came from his editorial and professorial attainments.

Of Samuel Gross, it is hardly necessary to speak, so widespread is his fame. He was Professor of Pathological Anatomy in the Cincinnati College for 5 years and Professor of Surgery in the University of Louisville for 16 years. He was a prolific contributor to the literature and a pioneer in operative surgery, being the first to suture a severed tendon. In 1880, with four others, including William Dawson, of Cincinnati, Gross originated the American Surgical Association and was also president of the American Medical Association.

Benjamin Winslow Dudley, known in his day as one of the greatest operating surgeons west of the Alleghenies, learned medicine in the same way that Daniel Drake had done—under a preceptor in his own town of Lexington, Ky. Sometime in 1810, he invested in a shipload of flour which he took to Spain to sell at wartime prices. From Spain, he went on to Paris, where he watched such surgeons as Dupuytren and Larrey, and then to London, where he was influenced by Abernethy and Astley Cooper. Dudley had a peculiarly retentive memory, and on his return to Lexington in 1814, he utilized this gift, as well as his sense of showmanship, to enhance his growing reputation as a skilful operator. He taught at Transylvania, where he came into conflict with Daniel Drake on one occasion, whereupon he challenged him to a duel. Drake declined the challenge, but his cause was taken up by one of his friends. Dudley wounded his opponent but then so skillfully dressed the wound that the man's life was saved and his friendship secured. Intensely jealous of his professional reputation, Dudley refused to allow the chairs of anatomy and surgery to be separated and held both of them throughout most of his life. Although best known for his skill in lithotomy, he was the first surgeon in

the United States to ligate the carotid artery for pulsating exophthalmos and the first one in the West to ligate the subclavian artery. He apparently disliked writing and left only fragmentary records, but, according to his pupils, he was emphatic on the subject of cleanliness, insistent about the use of boiled water for operative preparation, and extremely fond of the roller bandage. Despite the diminishing importance of Lexington as a medical center, he bitterly fought the proposal to transfer the Transylvania Medical Department to Louisville, and it was only with his retirement that Transylvania finally bowed to the inevitable.

On Christmas Day, 1809, another great Kentucky surgeon, Ephraim McDowell, performed the first ovariotomy, his patient being fully aware that such an attempt to cure ovarian disease had never previously been made. As McDowell worked, tying off the pedicle of a huge cystic structure and then incising the sac to evacuate some 15 lb. of material, his patient lustily sang hymns. The total weight of the tumor was 22½ lb.; the entire procedure took twenty-five minutes. By the fifth postoperative day, in an age when peritonitis was the dread accompaniment of even the simplest attempt at abdominal procedures, the patient was making her own bed, and on the twenty-fifth day she insisted on returning home to her hard pioneer life in the wilderness, sixty miles from Danville. Thus we see that successful early ambulation is far from being a modern discovery. McDowell did not report his case until he had operated successfully on two more women. His article, published in 1817 in a Pennsylvania journal, was generally ignored when its veracity was not actually questioned. His pupil, Alban Goldsmith, later a professor in the Medical College of Ohio, was in 1928 the second man to perform ovariotomy. Goldsmith also studied in Paris and was the first man in the United States to perform the operation of lithotripsy.

Another great American surgeon of the Ohio Valley was Reuben D. Mussey, a graduate of Dartmouth Medical School. It is his name which recurs throughout the ward books of the Cincinnati Commercial Hospital as having performed most of the operations done in that hospital. Mussey became Professor of Surgery in the Medical College of Ohio in 1838; in 1848, he reported sixteen operations performed with the use of chloroform "without unpleasant symptoms." In 1850, he became the fourth president of the American Medical Association. Gross records that Mussey was "painfully slow" as an operator but that his success was far greater than that of many of his more speedy colleagues. Mussey was particularly emphatic about preoperative and postoperative care. Gross rated him a dull lecturer but one whose lectures were valuable in "their practical adaptation to the daily and hourly wants of the practitioner."[3] Mussey, at a very early date, successively ligated both carotid arteries, allowing an interval of twelve days to elapse between ligations. In 1837, he was the second American surgeon to remove the scapula with a large portion of the clavicle, for osteosarcoma, in a patient who had previously had amputation at the shoulder joint. Mussey is said to have antedated Marion Sims in the successful surgical treatment of vesicovaginal fistula, and he performed forty-nine lithotomies, with only four deaths. He was, moreover, the first to show that bony union could occur in fractures of the neck of the humerus. He was the

3. Gross, S. D.: Autobiography of Samuel D. Gross, with Sketches of His Contemporaries, edited by his Sons, Philadelphia, G. Barrie, 1887, Vol. 2, p. 315.

avowed enemy of everything in the form of stimulants, and, like Drake, was a zealous lecturer on temperance. He was also a vegetarian who spent the later years of his life trying to establish vegetarianism on a scientific basis.

George Curtis Blackman supplemented his formal education in New York and London by serving as ship's surgeon on a private steamship line for five years, during which time he had leisure to read omnivorously. He always thought of himself as a Westerner and never failed to defend the surgical prowess of the West. He was a voluminous contributor to the periodical literature, as well as an editor and a translator of French works. Before Blackman came to the chair of surgery in the Medical College of Ohio, he had ligated both carotid arteries in one patient, resected the upper and lower jaws of another, had twice trephined the spine, and had successfully operated for aneurysm of the innominate artery. Before he was 35, he was acclaimed one of the most brilliant operative surgeons of his day. His operations covered the entire field of general surgery. He was at his best as a clinical teacher, having a profound distaste for the details of after-care. Like Drake, he was unable to adjust himself to giving didactic lectures to immature students. Juettner believed that "his wonderful control of the whole subject of surgery and the rapidity with which he would evolve principles and crystallize them into facts, drawing his illustrations and deductions from the whole domain of surgical science, made his lectures appear fragmentary and unsystematic." [4] He inspired great confidence and enthusiasm in the students who watched his operative demonstrations. Gross found him sensitive and irascible, a "rapid writer, a close thinker and an acute and bitter critic," but he also said: "Blackman wielded his scalpel with a bold and dashing hand. That he was at times reckless, those who knew him best did not hesitate to assert." [3] Blackman emerges from contemporary written accounts an erratic and difficult personality but one of the most brilliant operative surgeons of his time.

Most of these men, thus briefly discussed, had begun their careers before the time of anesthesia, and many of them never used anything more than brandy or opium with which to dull the sensibilities of their patients. They operated in their street clothes, occasionally using a protective apron for their own, rather than the patient's, sake. With the introduction of ether, surgery became less formidable; the surgeon could attend more carefully to hemostasis and specific pathology when he no longer needed to work at top speed on a tense, suffering, and often writhing patient.

William Morton patented his "letheon" device for vaporizing ether in 1846, and its use in the Cincinnati Commercial Hospital is recorded as early as March, 1847, though at least one patient, exhilarated by his first whiff, refused to inhale any more. Thus the complexion of surgery was markedly altered, and it was to be further altered, if not revolutionized, by the bacteriological discoveries of Pasteur, the antiseptic theories of Lister, and the cellular pathology of Virchow. With the tools derived from all this knowledge, surgery became a specialty worthy of the name, and American surgeons advanced rapidly in their contributions to the old art, which now became a new science.

4. Juettner, O.: Daniel Drake and His Followers: Historical and Biographical Sketches, 1789-1909, Cincinnati, Harvey Publishing Co., 1909, p. 231.

As medicine and surgery became established on a more scientific basis, boards of health with legal powers arose; medical organizations became more compactly organized, placing greater emphasis on communicating individual advances and achievements and sponsoring periodicals. It was not until 1861 that the first nephrectomy was done by Charles Stoddard, of Wisconsin. In 1867, John Bobbs, of Indianapolis, under the impression that he was dealing with an ovarian cyst, performed the first choledochostomy, and eleven years later, Marion Sims diagnosed and deliberately drained a distended gall bladder. Other surgical innovations came still later; it was 1889 when William S. Halsted devised his curative operation for inguinal hernia. Although most of the major "firsts" in surgery came after the Civil War, the scientific advances which made them possible had already existed before the war. Scientific progress on an increasingly specialized basis was rapid. In the century ending in 1851, surgery was almost uniformly empirical, being carried on by a few great pioneers who stand out above the general picture of primitive conditions and inadequate knowledge and whose success lay chiefly in a combination of daring and good luck. Between 1851 and the present, there has been a prodigious advance in surgical knowledge and techniques, the germ of which upsurge was already planted by 1860. With the subsequent great advances in the field of American surgery, all are undoubtedly familiar.

To sum up briefly, during the first period of development, that is, 1788 to 1815, American surgery was truly in a pioneer stage. The way of progress was unblazed, and the surgical frontiers were unlimited, literally and figuratively. From 1815 to 1846, surgery developed along with the rising settlements, and recognized medical schools and techniques arose. The surgical frontiers were therefore necessarily somewhat narrowed, but the primary emphasis remained democratic and empirical. As one frontier became fixed, another opened. Experimental work began to give a scientific basis to medical and surgical therapy. By 1860, trends toward specialism were evident. In 1876, Gross wrote: "It is safe to affirm that there is not a medical man on this continent who devotes himself exclusively to the practice of surgery," [5] yet Gross himself, as well as Valentine Mott, Willard Parker, and Benjamin Dudley, were even then recognized primarily as surgeons.

By 1860, the patterns were formed; frontiers ceased to narrow and began once again to expand. Fields hitherto closed were opened, and, if the individual was no longer a pioneer in the literal sense, he became one in the more restricted field of science. Today, well-known surgical adjuncts open previously impenetrable frontiers, and it is no longer as an unarmed empiric that the surgeon passes each new boundary. Rather, the pioneer has given way to the investigator, who bases considered risks on the reasoning acquired from prior knowledge and who fulfills Samuel Gross's patriotic reply to a European detractor:

> The American mind is the most active mind in the world; and if the American physician is not as highly educated as the English, French, and German—if he does not know as much Greek and Latin, mathematics, astronomy, and the more abstruse branches of a university education—he may justly pride himself upon his superiority of common sense, of Yankee ingenuity and smartness . . .[6]

5. Gross, S. D.: Surgery, in A Century of American Medicine, 1776-1876, Philadelphia, Lea, 1876, p. 117.

6. Gross, S. D.: American Versus European Medical Science, Med. Rec. **4**:190, 1869.

CRITICAL EVALUATION OF JEJUNOSTOMY

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THE RESTORATION and maintenance of an adequate nutritional status in patients unable to eat by mouth continue to be trying problems despite the emphasis that has been placed on the importance of nutrition in the surgical literature during recent years.[1] Ordinarily in such situations intravenous administration of the required fluids is practiced, though this method has serious shortcomings when its use is necessary for more than a few days. These disadvantages include the danger of overloading the circulation in cardiac patients, prolonged periods of immobilization, and the difficulty of providing adequate amounts of calories and protein. In some situations feeding by means of a nasogastric tube has been attended by excellent results, and this method is particularly useful when a small caliber polyethylene tube is employed in conjunction with a force pump. However, this method in certain instances is impracticable because of such factors as mechanical obstruction to the passage of a tube or the presence of an anastomotic suture line. In such circumstances the use of jejunostomy may wisely be considered.[2] Case and his associates have established two principles of fundamental importance in the consideration of jejunostomy: (1) the practicability of homogenized milk as the basis for a well-tolerated feeding formula, and (2) the safety and freedom

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1. Jones, C. M., and Eaton, F. B.: Postoperative Nutritional Edema, Arch. Surg. **27**:159 (July) 1933. Ravdin, I. S.; Stengel, A., Jr., and Prushankin, M.: The Control of Hypoproteinemia in Surgical Patients, J. A. M. A. **114**:107 (Jan. 13) 1940. Mulholland, J. H.; Tui, C.; Wright, A. M., and Vinci, V. J.: Nitrogen Metabolism, Caloric Intake and Weight Loss in Postoperative Convalescence, Ann. Surg. **117**:512 (April) 1943. Elman, R.: Acute Starvation Following Operation or Injury, with Special Reference to Caloric and Protein Needs, ibid. **120**:350 (Sept.) 1944. Varco, R. L.: Preoperative Dietary Management for Surgical Patients, Surgery **19**:303 (March) 1946.

2. Wolfer, J. A.: Jejunostomy with Jejunal Alimentation, Ann. Surg. **101**:708 (Feb.) 1935. Clute, H. M., and Bell, L. M.: Jejunostomy for Postoperative Feeding, ibid. **114**:462 (Sept.) 1941. Colp, R., and Druckerman, L. J.: The Indications for Jejunal Alimentation in the Surgery of Peptic Ulcer, ibid. **117**:387 (March) 1943. Rosenak, S., and Hollander, F.: Surgical Jejunostomy for Alimentation, Clinics **3**:638 (Oct.) 1944.

from complications of the Stamm technique.[3] With these principles in mind, we have reviewed our experiences with 103 jejunostomies over the last four years in order to determine the nutritional efficiency of jejunostomy feedings, the principles of administration of such feedings, and the indications that may exist for the use of this procedure.

COMPOSITION OF THE FEEDING FORMULA

Jejunal feeding mixtures proposed in past years, excellent from the standpoint of nutrition but very complex in their formulation, have not achieved widespread use.[4] We have continued to use homogenized milk as the basic ingredient in the feeding formula. Milk, obviously, is a readily available, economical, and nutritionally complete food, but whole milk introduced into the jejunum results all too frequently in cramps and diarrhea. Homogenized milk, because of the minuteness of its fat particles, to a large extent is free of these obligations, being well tolerated in about 85% of cases. Worthy of mention is the fact that commercially canned condensed milk is homogenized and when reconstituted with water may be substituted for fresh homogenized milk with equal effectiveness.[5]

This basic formula may be enriched by the addition of protein, carbohydrate, vitamins, potassium salts, and bile to meet situations in which these additional ingredients are indicated. Homogenized milk contains approximately 35 gm. of protein and 700 cal. per 1,000 cc. To build up badly depleted patients it is desirable to use a formula more concentrated in both calories and protein, and to this end we have added to 1,000 cc. as much as 60 gm. of starch hydrolysate [6] and 50 gm. of protein hydrolysate [7] over a period of days. The latter mixture contains about 72 gm. of protein and 1,090 cal. per 1,000 cc.

Although 2,000 cc. of homogenized milk daily more than meets the basic body requirements for potassium, inasmuch as there are about 35 mEq. of potassium per 1,000 cc. of milk, in situations in which the losses of potassium from the body are excessive or in which a potassium deficit already exists additional potassium may be readily supplied by jejunostomy as a prophylactic or therapeutic measure. We have used a salt mixture containing 1 gm. each of potassium citrate, potassium acetate, and potassium bicarbonate dissolved in 8 cc. of water, as originally described by Randall and co-workers.[8] Three grams of this preparation (27 mEq. of potassium) given three or four times a day is usually adequate to replace all but the most abnormal losses and is well tolerated by the jejunum.

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3. Case, C. T.; Zollinger, R. M.; McMullen, C. H., and Brown, J. B.: Observations in Jejunal Alimentation, Surgery **26**:364 (Sept.) 1949.

4. Scott, H. G., and Ivy, A. C.: Jejunal Alimentation, Ann. Surg. **93**:1197 (June) 1931. Hollander, F., and Sober, H. A.: A Modified Synthetic Predigested Aliment for Protracted Jejunostomy Feeding, Surgery **25**:500 (April) 1949.

5. Poth, E.: Personal communication to the authors.

6. Dexin, brand of starch hydrolysate, manufactured by Burroughs Wellcome & Company, Inc., Tuckahoe, N. Y.

7. Protolysate, brand of protein hydrolysate, manufactured by Mead Johnson & Company, Evansville, Ind.

8. Randall, H. T.; Habif, D. V.; Lockwood, J. S., and Werner, S. C.: Potassium Deficiency in Surgical Patients, Surgery **26**:341 (Sept.) 1949.

Similarly, in those instances of external biliary fistula wherein bile is drained to the outside, this bile may be collected and refed in the jejunostomy, with benefit to the patient. In appropriate situations we have mixed with the feeding formula as much as 500 cc. of bile daily.

The requirements of a satisfactory jejunostomy feeding formula, as advanced by Stewart and his associates, are well fulfilled by the above described nutrients.[9]

METABOLIC STUDIES

The nutritional effectiveness of the homogenized milk formula was carefully evaluated by nitrogen balance studies together with observations of the weight trends and the total circulating protein. Eleven such metabolic studies were carried out under closely controlled conditions. The total daily volume of the feedings was maintained at 2,400 cc. given in 10 equal feedings at two-hour intervals. Repeated studies during periods of three days in which only homogenized milk was used were

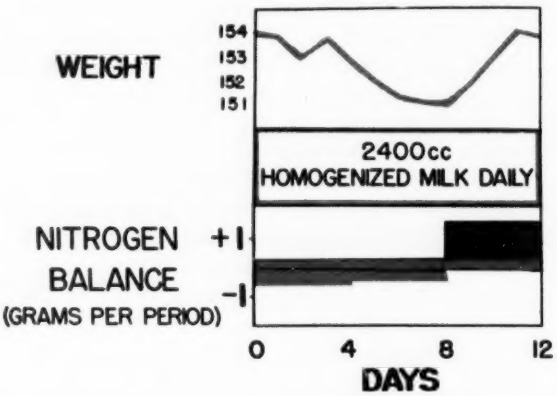

Chart 1.—Relatively constant weight and nitrogen balance equilibrium maintained by homogenized milk alone in Patient J. E. with a choledochostomy.

run in each instance. The results of these studies demonstrated that in most instances nitrogen balance could be maintained with the basic homogenized milk formula, which provided 1,680 cal. and 84 gm. of protein per 2,400 cc. (Chart 1). It should be noted, however, that in those cases in which the basic disease was an advanced carcinoma of the upper gastrointestinal tract or pancreas, it was not possible by these means to achieve a positive nitrogen balance but only to hold to a minimum the magnitude of the negative balance.

To evaluate further the efficiency of the jejunostomy, fat balance studies were carried out in order to determine the completeness of the fat absorption. This was deemed a necessary part of the investigation, since the 4% fat in homogenized milk makes up about half the caloric value and since jejunostomy feedings bypass the gastric and duodenal phases of digestion. The subjects for these tests received fixed amounts of fat in the form of homogenized milk for periods of three to six

9. Stewart, J. D.; Hale, H. W., Jr., and Schaer, S. M.: Management of Protein Deficiency in Surgical Patients, J. A. M. A. **136**:1017 (April) 1948.

days. Analyses of the fecal fat during these periods were carried out. It has been shown previously that normal persons absorb 95% or more of all fat ingested by mouth. Absorption of fat from jejunal feedings in these studies varied from 65 to 82% of normal, demonstrating a significant deficiency in this regard.

The work of Jones and others, suggested the possibility of improving the fat absorption by adding an emulsifying agent to the formula.[10] Sorethytan (20) monooleate ("tween 80"; sorlate®) has been shown to be an effective emulsifying agent, and fat balance studies were repeated with the addition of this compound to the homogenized milk. The percentage of fat absorption in these repeated studies was close to normal, as illustrated in Chart 2. Because of this striking improvement, 1 cc. of sorethytan (20) monooleate is added as a routine measure to each 1,000 cc. of homogenized milk used for jejunostomy feedings, and, as a result, about 200 to 250 cal. per 2,400 cc. of homogenized milk, otherwise lost, is retained by the body for effective utilization.

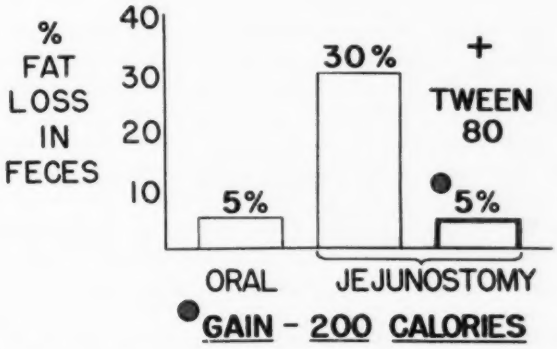

Chart 2.—Fat balance study. Improvement in fat absorption by jejunostomy with the addition of an emulsifying agent, sorethytan (20) monooleate ("tween 80").

Attempts were also made to increase the caloric value of the formula by increasing the percentage of fat. Stable mixtures of corn oil in water and milk with added butterfat were prepared, with fat concentrations varying between 6 and 12%. Sorethytan (20) monooleate was added to all these mixtures. A few patients were able to tolerate 8% fat mixtures for a few days without cramps or diarrhea, but most of our patients were unable to tolerate even the 6% fat mixture for a prolonged period. Bulky fatty stools and diarrhea customarily developed after the patients had been about 48 hours on these feedings. More recently, we have tried a mixture of vegetable oil and dextrose[11] (lipomul-oral) prepared in a stable homogenized form. The concentration of fat used with this preparation varied between 6 and 10%, and again concentrations of more than 6% were not tolerated, and, for the most part, even 6% fat mixtures produced fullness, cramps, and

10. Jones, C. M.; Culver, P. J.; Drummey, G. D., and Ryan, A. E.: Modification of Fat Absorption in the Digestive Tract by the Use of an Emulsifying Agent, Ann. Int. Med. **29**:1 (July) 1948.

11. Lipomul-oral was supplied by the Upjohn Company, Kalamazoo, Mich.

frequency of defecation. The increased intestinal motility and the failure to utilize the added calories have made these higher fat mixtures impracticable for jejunostomy feedings.

The addition of supplementary protein and carbohydrate to the homogenized milk may be accomplished, as mentioned previously, to enrich the caloric and protein values of the formulas. The value of such enrichment is greatly limited, however, by the fact that an interval of 7 to 10 days after establishing the jejunostomy is usually required before they are well tolerated. With toleration established, the addition of up to 60 gm. of starch hydrolysate and 50 gm. of protein hydrolysate to 1,000 cc. of homogenized milk provides a formula with which large positive nitrogen balances may be achieved.

REGULATION OF FEEDING PROGRAM

In the regulation of a jejunostomy feeding program there is a high degree of individual variation, and it must be accepted that considerable time and effort are necessary at times to obtain consistently good results. The value of a jejunostomy may in large part be lost if these factors are not given due regard, and certainly they mitigate against its use in any sense as a routine measure. Customarily, no use is made of the jejunostomy for 12 to 18 hours after its creation; during this time the jejunostomy tube either may be clamped or connected to a system for siphonage drainage to a bottle beside the patient's bed.[12] After this interval, the administration of water and electrolytes is begun, starting with 50 cc. of 5% dextrose in water introduced by an asepto syringe at hourly intervals. The use of a Murphy drip apparatus may be of some advantage, but its proper regulation is a constant chore. By the second day of its use, 100 cc. of solution hourly will ordinarily be well tolerated. As a general rule, about 1,000 cc. of fluid may be given by this route during the first day and from 1,500 to 2,000 cc. on the second.

Because distention may be a factor interfering with the early efficient function of a jejunostomy, we have used during the first 24 to 72 hours rather liberal amounts of liquid petrolatum as an aid in promoting peristalsis and have introduced into the jejunum as much as 30 cc. repeated at 6- or 8-hour intervals over the course of a 24 hour period. Only after normal bowel activity is restored are homogenized milk feedings started. Fifty cubic centimeters of milk hourly is the initial ration, and this is gradually increased in the following one to three days to 200 cc. every two hours. Appropriate amounts of water and electrolytes are continued during this time.

For the ordinary short-term jejunostomy feeding program, lasting not more than two weeks, homogenized milk alone provides an adequate source of fluid, electrolytes, and nutrition. Of course, additional water, electrolytes, and vitamins may be added in amounts deemed necessary. A daily intake of 2,000 cc. of homogenized milk provides approximately 1,400 cal. and 70 gm. of protein. In those situations in which large nutritional losses must be replenished and in which jejunostomy feedings are to be continued over a more prolonged period, the feeding formula may be enriched by the addition of more carbohydrate and more protein, as previously described. Such enrichment may ordinarily be started only after

12. Rosenak, S., and Hollander, F.: Early Postoperative Motor Response of the Small Intestine to Jejunal Feedings, S. Clin. North America **27**:345 (April) 1947.

a lag of 7 to 10 days after construction of the jejunostomy. Thirty grams of starch hydrolysate and 50 gm. of protein hydrolysate are added to 1,000 cc. of homogenized milk and the same volume of feeding continued. This provides approximately 1,000 cal. and 70 gm. of protein per 1,000 cc. of feeding. If this formula is well tolerated, the amount of starch hydrolysate may be increased to 60 gm., thus providing about 1,100 cal. per 1,000 cc. This last formula is as concentrated as can ordinarily be tolerated. These enriched formulas make possible the correction of major nutritional defects and permit a rapid gain in weight (Chart 3).

As a result of the unpredictable factor of individual tolerance to this program, a few guides have been developed to smooth out the process of adjustment. Distention produced by the feeding should be avoided, and regular observation of the patient is essential in this regard. When advances in volume or caloric concentration of the feedings are made, the patient must be evaluated at intervals to determine his tolerance for the new feedings. If distention or cramps should occur, the feedings are discontinued for a time, and the tube may be used for decompression.

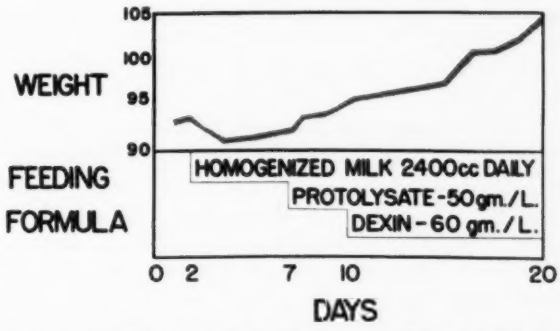

Chart 3.—Chart showing rapid gain in weight made possible by enriched feeding formula. Patient H. C. had carcinoma of the stomach resulting in 40 lb. (18 kg.) weight loss.

The feedings are best discontinued between midnight and 6 a. m. in order to give the patient and the intestinal tract a rest.

In our experience the effectiveness of the early use of the jejunostomy depends on the return of normal peristalsis. The use of liquid petrolatum has seemed to be of real value in hastening this return. Distention, rather than diarrhea, is more frequently the problem to be averted or overcome. Diarrhea, however, will occur if milk is added to the program in large amounts during the period of ileus.

As the convalescence of a patient progresses and he is able to start on oral feedings, the amount of feeding per jejunostomy is accordingly reduced. When the patient is fully able to meet his fluid and nutritional needs by mouth, the jejunostomy tube is removed. A small amount of drainage usually persists from the sinus tract for a few days, but the opening has sealed itself promptly in our cases, with rare exception.

TECHNIQUE OF JEJUNOSTOMY

The Stamm technique has been employed in every instance in our series. A No. 16 French catheter is introduced into the jejunum at a point of the antimesenteric border just far enough below the ligament of Treitz to permit easy approximation of the jejunum to the anterior

abdominal wall. The catheter is passed distalward into the lumen of the bowel for 10 to 15 cm. and is then secured in place with two concentric purse-string sutures of fine silk. The outermost of these sutures includes a bit of the catheter. The proximal portion of the catheter is then drawn out through a small stab wound in the left epigastrium, and the loop of jejunum is drawn up flush with the abdominal wall. This portion of jejunum is anchored in place to the peritoneum with a few interrupted sutures in such fashion as to insure against angulation at the site of entrance of the catheter. We prefer this simple technique to the Witzel procedure, since use of the latter method, whether by infolding of bowel wall or by angulation, will occasionally result in obstruction.

EXPERIENCE AND RESULTS

During the past four years, 103 jejunostomies have been performed on the general surgical service at the University Hospital. Of these, 85 have been complementary procedures performed at the time of a primary operation. In seven instances it was employed as a preliminary procedure to better prepare these patients for definitive surgery. Four of the jejunostomies were created in the postoperative period, and, finally, seven were done as primary procedures. In the latter cases the jejunostomy was performed as a palliative measure when gastrostomy or gastroenterostomy was not feasible. We will not attempt in this discussion to evaluate the procedure for palliation.

Operations on the stomach were the most frequent in which jejunostomies were used as adjuncts, numbering 76. In 12 cases the primary operation was on the biliary tract. Operations involving the pancreas, duodenum, and esophagus made up the remainder. In the great majority of these cases significant weight loss had occurred prior to the establishment of the jejunostomy, and this was of a marked degree in 50 patients, varying between 20 and 66 lb. (9.1 and 29.9 kg.).

The records of these 103 cases were carefully reviewed as objectively as possible with respect to an over-all evaluation of the jejunostomy procedure. In 27 instances the procedure was deemed to have great value. In this group there was marked improvement in the patient's status, and his care was greatly simplified. In a few cases, difficult to assess in precise figures, the jejunostomy seemed to be of such value as to be virtually lifesaving. The factors to be dealt with in the latter situations included progressive loss of weight with marked debility, draining biliary fistulae with long-standing jaundice, and prolonged postoperative obstruction at a gastrojejunal stoma. In 56 cases the jejunostomies were classified as definitely worth while. In this group the procedure contributed to the improvement of the patients and to their ease of management but was scarcely indispensable. In 20 patients the jejunostomy had little or no value.

In 88 of the 103 cases the tube feedings, both water and later homogenized milk, were tolerated well. However, in many of these cases, adjustments in volume or in concentration of the feedings were needed occasionally to control abdominal cramps, mild distention, and, infrequently, diarrhea. These adjustments did not appreciably impair the efficiency of the jejunostomy in this group. In the remaining 15 cases the tube feedings were poorly tolerated so that the jejunostomy had little or no value. Distention and abdominal cramps were produced by the feedings in these patients. In at least half these cases, toleration to the feedings could probably have been established if proper initial regulation of the feeding program had been attempted. In the remainder, however, toleration to homogenized milk simply could not be attained despite repeated and careful manipulation of the feeding

program. Hence, in this series, for reasons either of patient intolerance or of improper management, in about 15% of the cases a high caloric feeding program could not be tolerated.

As mentioned previously, in our over-all evaluation of this series, we considered the jejunostomy to be of little or no value in 20 cases. Fifteen of these are accounted for in the group that did not tolerate the homogenized milk formula. Three of the remaining five died of the magnitude and severity of their disease before the jejunostomy could possibly be evaluated. Finally, in two cases the jejunostomy served little purpose because the patients were able to take oral fluids and nutrition so well within three or four days after surgery.

No serious complications have resulted from the jejunostomies in this series. We have seen no obstructions either at the jejunostomy site or from volvulus or some other mechanism secondary to the procedure, nor have there been any cases of peritonitis or intra-abdominal abscesses as a consequence of a jejunostomy. Frequently, mild induration and redness occur around the stab wound where the jejunostomy tube is anchored, but no suppurative wound infections have developed. The tubes were left in place for periods varying between 4 and 110 days, although in most instances they were removed by the 10th postoperative day. After the tube is withdrawn there is usually slight drainage onto the skin from the artificially created fistula, but this customarily seals off completely within 48 hours. In six cases this drainage persisted for longer periods up to a week, but it has always been rather slight. Some excoriation of the skin may occur with this drainage if the skin is not properly protected.

COMMENT

During the time that experience with the homogenized milk formula and management of a feeding program was being developed and confidence in the technical procedure gained, we used jejunostomies rather freely and without fixed indications. On the basis of this experience the procedure is now limited to well-defined indications. The patients for whom jejunostomy offers the most advantage fall into two general groups, namely, those who have lost major amounts of weight (more than 20 lbs.), and those who fall into the elderly, poor-risk cardiac classification. Whether the jejunostomy should be performed as a preliminary or as a complementary procedure depends largely on the nutritional status of the patient when first seen.

With respect to the definitive operations in conjunction with which jejunostomy should be employed, extensive gastrectomies and complicated biliary tract or pancreatic operations have derived the most benefit. In the latter it is of distinct advantage to be able to feed back into the intestinal tract bile draining from an artificially created fistula. This is especially true in patients who have been jaundiced for a long time because of a stricture of the common bile duct or because of an obstructing neoplasm. In a rare instance the procedure has been valuable in the postoperative course to meet the complication of malfunctioning gastrojejunal stoma.[13]

13. Allen, A. W., and Welch, C. E.: Jejunostomy for the Relief of Malfunctioning Gastroenterostomy Stoma, Surgery **9**:163 (Feb.) 1941. Walters, W., and Hartman, H. R.: Preoperative and Postoperative Care of Patients with Lesions of the Stomach and of the Duodenum, Arch. Surg. **40**:1063 (June) 1940.

SUMMARY

1. The indications for jejunostomy are relatively uncommon, but in appropriate situations its use is often of great value.

2. A formula with homogenized milk as its basic ingredient is well tolerated in about 85% of the cases.

3. This formula may be enriched with protein hydrolysate, carbohydrate hydrolysate, vitamins, bile, and potassium when these are indicated. The addition of an emulsifying agent, sorethytan (20) monooleate ("tween 80") improves the efficiency of the fat absorption.

4. Jejunostomy is advocated in patients who have suffered marked loss in weight or who fall into the elderly poor-risk cardiac classification in whom such procedures as extensive gastrectomy or complicated biliary tract and pancreatic operations are needed.

5. The procedure should also be kept in mind as a sound method for handling the postoperative complication of a prolonged malfunctioning gastrojejunal stoma.

MAYDL JEJUNOSTOMY

Technical and Metabolic Considerations

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KATE DAUM, Ph.D.
AND
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IN SPITE of a more aggressive approach in the treatment of malignant disease of the upper intestinal tract that has come about during the past few years, there still remains a large group of unfortunate people on whom even palliative extirpation of their lesions cannot be safely performed. We still encounter, therefore, as a result of obstruction, pain, or persistent fistulae, situations in which for a considerable length of time feeding through an artificial stoma must be utilized.

Our experience with permanent gastrostomy at such times has not been too rewarding. In spite of the many techniques available, sooner or later most gastrostomy stomas leak or develop an unpleasant odor. The frequency of feeding becomes a nuisance, not only to the patient but to his entire household. This has led us to reexplore the efficacy of jejunal feedings when such feedings are required.

Of the four principal varieties of jejunostomy [1] (Fig. 1) only the von Eiselsberg, which applies Witzel's gastrostomy principle to jejunostomy, and the Maydl remain in general use at present.

The Maydl jejunostomy has been utilized by us on 34 occasions during the past four years. It has proved to be an unusually satisfactory feeding fistula. This jejunostomy is particularly useful in situations in which such a feeding fistula is likely to be required for many weeks or months, either constantly or intermittently. In contrast, the von Eiselsberg (Witzel) jejunostomy is ideally adapted to patients in whom only temporary jejunal feeding is desired. The Maydl jejunostomy has its greatest application in patients for whom oral feedings are not possible because

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From the Departments of Surgery and Nutrition, State University of Iowa College of Medicine, and the Department of Surgery, University of North Carolina School of Medicine, Chapel Hill, N. C.

1. Surmay: De l'entérostomie, Bull. gen. therap. med. **94**:445-45, 1878; Observation d'entérostomie, ibid. **95**:198-208, 1878. Maydl, K.: Über eine neue Methode zur Aüsfuhrung der Jejunostomie und Gastroenterostomie, Wien. med. Wchnschr. **42**:740-743, 1892. Albert E.: Eine neue Methode der Jejunostomie, ibid. **44**:57-59, 1894. Witzel, O.: I. Zur Technik der Magenfistelanlegung, Zentralbl. Chir. **18**:601-604, 1891. von Eiselsberg, A. F.: Über Ausschaltung inoperabler Pylorus-Stricturen nebst Bemerkungen über die Jejunostomie, Arch. klin. Chir. **50**:919-939, 1895.

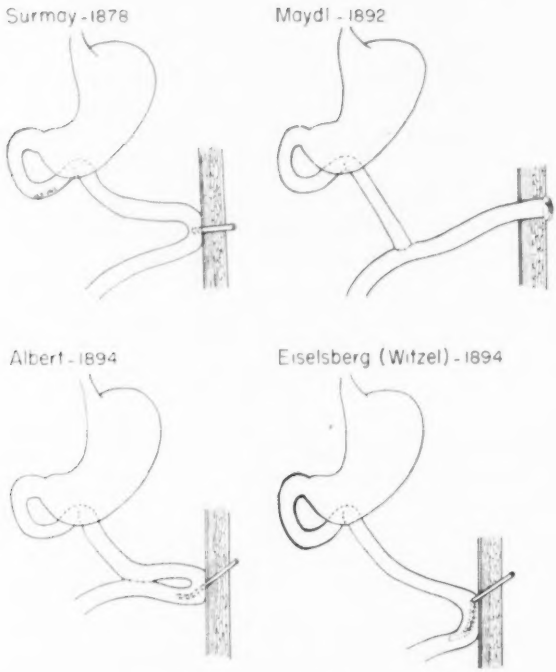

Fig. 1.—Chief types of jejunostomy.

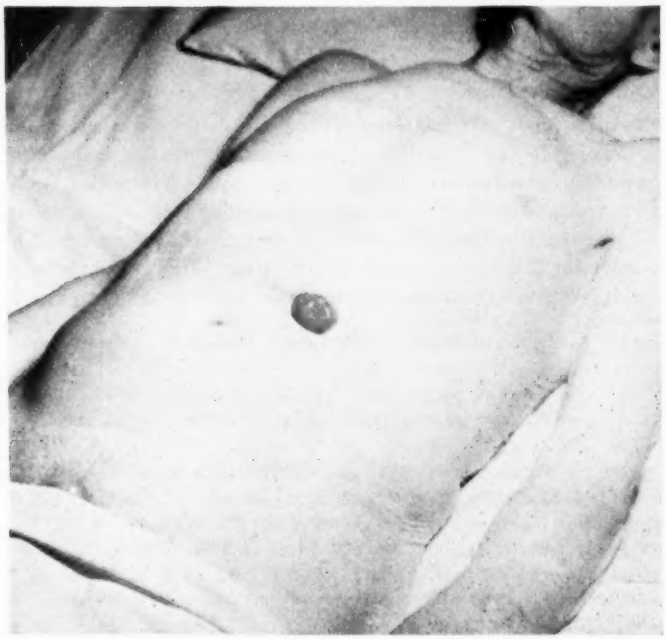

Fig. 2 (Patient M. L.).—Photograph showing incision and abdominal stoma 19 days after establishment of Maydl jejunostomy.

of obstruction of the pharynx, esophagus, or stomach by inoperable carcinoma. We prefer its use in place of permanent gastrostomy in instances in which either would provide a feeding fistula distal to an obstruction.

The Maydl jejunostomy provides the following advantages:

1. A large feeding stoma is provided through which thick formulae can be administered, with the use of a large bore rubber feeding tube.

2. No indwelling rubber tube is required. Between feedings a small gauze pad is applied over the enterostomy.

3. There is no tendency for spontaneous and premature healing of the fistula.

4. The procedure does not involve infolding of the jejunal wall, and hence no narrowing of bowel lumen occurs.

5. With minimal instruction the patient and family view the feeding procedure with less trepidation than if an indwelling tube is present.

6. The area is odorless, and the surrounding skin is not irritated. Leakage of bile and pancreatic juice and regurgitation of feedings do not occur if the technical procedure is properly carried out. Such leakage is often seen in the permanent gastrostomy or the von Eiselsberg jejunostomy.

7. Large feedings administered at infrequent intervals are well tolerated.

8. When a patient is able to take a small amount of food by mouth, supplemental feeding is much easier through the use of the Maydl jejunostomy.

9. Food intake can be regulated to such an extent that weight gain has been frequently encountered.

OPERATIVE TECHNIQUE

This operation is performed employing either general or local anesthesia. A short transverse incision is made in the lower portion of the left upper quadrant of the abdomen. The ligament of Treitz is the guide to the proximal jejunum. The jejunum and mesentery are divided about 8 to 10 in. (20.3 to 25.4 cm.) below the ligament of Treitz. The end of the proximal jejunum is anastomosed to the side of the distal jejunum 18 to 20 in. (45.7 to 50.8 cm.) distal to the site of jejunal division. The mesentery is closed to prevent leaving an aperture. The end of the distal jejunal segment is brought out through the operative incision or a separate incision as a terminal enterostomy. The divided mesentery of the distal jejunal segment is sutured to the parietal peritoneum of the left colic gutter to cover raw mesenteric margin, to close the aperture which would otherwise remain in this area, and to fix the bowel intraperitoneally to prevent postoperative prolapse. After the incision is closed, the bowel margin is sutured to skin margins to insure prompt mucosa-to-skin healing, thus lessening the possibility of excessive scar formation with subsequent stomal stenosis.

CLINICAL OBSERVATIONS

In this series of 34 Maydl jejunostomies, there was in no instance obstruction, leakage, or other complication at the site of the end-to-side jejunostomy.

Serious complications occurred early in this series in two patients when "abdominal limbs" of jejunum of only 6 and 10 in. (15.2 and 25.4 cm.), respectively, in length were formed. In these patients severe leakage of bile and pancreatic juice resulted in painful digestion of skin surrounding the stoma. In the first of these two patients in order to aid skin healing a balloon catheter of the Foley type was inserted into the 6 in. abdominal (jejunal) limb, and the inflated balloon was drawn

snugly against the abdominal wall. Drainage and regurgitation were prevented, but the pressure caused necrosis of jejunum and fatal peritonitis ensued. In the second patient, regurgitation from the 10-in. jejunal limb was so frequent and severe that the jejunostomy was revised. In other instances the end-to-side implantation was located from 15 to 24 in. (38.1 to 60.9 cm.) from the abdominal stoma, the usual distance being about 18 in. In these patients there was either no reflux of intestinal contents or injected formula from the stoma, or there was occasional and minimal reflux in only the early postoperative period. Possibly reverse peristalsis or ileus accounted for this transitory phase. Apparently normal peristalsis is effective in preventing stomal drainage in such a jejunal limb, and the formation of a valve is not necessary. Feedings of 250 cc. were retained, and in many patients rapid instillation of 500 cc. or more was possible without regurgitation or apparent ill effects.

Jejunostomy feedings are usually begun the day following operation. Initially, small feedings (30 to 60 cc.) of glucose water or skimmed milk are injected at frequent intervals. Later, larger feedings (250 to 350 cc.) of gastrostomy-type formula are utilized. The formula used is basically milk but is fortified by the addition of supplemental protein foods or amino acids, sugar, and vitamins. In some instances diarrhea occurs. The diarrhea can be controlled by reducing either the amount of fat or of sugar (or both) in the formula. No patient had diarrhea while receiving skimmed milk with supplemental amino acids as his jejunostomy formula. All patients who had diarrhea immediately postoperatively, when given the usual jejunostomy feedings gradually became able to tolerate full feedings without diarrhea.

In five patients jejunostomy prolapse occurred several weeks or months after operation. In these instances, fixation of the mesentery had not been carried out. Manual reduction of the prolapse was successful in four patients, but in the fifth, operative correction was necessary.

In seven patients, glucose absorption was studied. Blood glucose determinations were made at half-hour intervals after the instillation of 50 gm. of glucose into the abdominal jejunal limb, first in the fasting state and secondly two hours later. Hyperglycemic and hypoglycemic blood sugar levels were frequently found. Despite this, in none of the patients studied did the symptoms said to be characteristic of the Zollinger-Hoerr syndrome develop.[2] The frequent occurrence of significant hyperglycemic levels, after the administration of glucose into the jejunum, without the development of clinical symptoms, suggests that in the postgastrectomy Zollinger-Hoerr syndrome the blood level is not the only factor involved. Furthermore, sudden distention of the jejunum seemed to be no factor, nor did the association of jejunitis. From this experience we must raise some question regarding the accuracy of the term "dumping syndrome" in the description of this odd group of symptoms.

Food utilization studies were carried out on six patients following Maydl jejunostomies. During a period of weighed food intake, five-day collections of

2. Zollinger, R. M., and Hoerr, S. O.: Gastric Operations: Troublesome Postoperative Symptoms with Special References to Carbohydrate Ingestion, J. A. M. A. **134**:575-579 (June 14) 1947.

urine and stools were made and nitrogen, calcium, phosphorus, and iron excretions determined. The methods used for analysis were as follows:

Nitrogen.....Macro-Kjeldahl [3]
Calcium.....McCrudden [3]
Phosphorus....Fiske and Subbarow [3]
Fat.....Soxhlet extraction [3]
Iron.....Fortune and Mellon [4]

Tables 1 and 2 summarize the results on these six patients. Two months after the operation two periods were run on Patient 1. In all patients positive nitrogen, calcium, phosphorus, and iron balances were established. Only one patient showed

TABLE 1.—*Daily Nitrogen and Fat Utilization (Average of Five-Day Periods)*

Patient	Time After Operation, Days	Nitrogen Intake, Gm.	Nitrogen Output			Caloric Intake	Fat	
			Urine, Gm.	Fecal, Gm.	Balance, Gm.		Intake, Gm.	Output Fecal, Gm.
1.....	7	12.1	13.07	1.34	−2.31	1,948	50.6	8.61
1.....	60	12.1	10.11	1.29	+0.70	2,049	52.1	3.08
1*.....	65	16.2	13.42	1.22	+1.56	3,023	72.5	3.61
2.....	7	12.8	9.93	1.28	+1.59	2,152	78.1	6.42
3.....	14	17.0	Total	16.1	+0.90	2,514	66.9	4.89
4†.....	21	18.5	10.66	6.04	+1.80	1,947	47.8	6.71
5‡.....	14	29.5	24.17	5.13	+0.20	1,907	36.1	5.49
6‡.....	3	28.04	Total	27.76	+0.28	2,430	....	....

* Patient was able to take food by mouth in six feedings.
† Alacta® (powdered whole milk) was used in formula.
‡ Casein hydrolysate was used either in formula or intravenously.

TABLE 2.—*Daily Calcium, Phosphorus, and Iron Utilization (Average of Five-Day Periods)*

Patient	Time After Operation, Days	Calcium				Phosphorus				Iron			
		Intake, Gm.	Urine, Gm.	Fecal, Gm.	Balance, Gm.	Intake, Gm.	Urine, Gm.	Fecal, Gm.	Balance, Gm.	Intake, Mg.	Urine, Mg.	Fecal, Mg.	Balance, Mg.
1	7	1.700	0.229	1.467	+0.004	1.593	0.769	0.719	+0.105	12.54	0.120	11.540	+0.88
1	60	2.311	0.237	1.845	+0.229	2.184	0.928	0.898	+0.358	.....	.....	.....	.....
1*	65	2.044	0.233	1.207	+0.604	2.590	1.071	0.622	+0.897	.....	.....	.....	.....
2	7	2.008	0.162	1.195	+0.651	2.284	1.023	0.643	+0.618	10.72	0.420	9.080	+1.22
3	14	2.427	Total	2.001	+0.426	2.569	Total	1.646	+0.923	12.36	Total	11.905	+0.455
4†	21	2.017	0.264	1.494	+0.259	2.591	1.153	.963	+0.475	11.50	0.314	10.066	+1.120
5‡	14	1.727	0.141	1.508	+0.078	1.891	0.941	.801	+0.149	7.71	0.121	7.444	+0.145
6‡	3	1.614	Total	1.512	+0.102	1.789	Total	1.511	+0.278	8.82	Total	8.410	+0.410

* Patient was able to take food by mouth in six feedings.
† Alacta® (powdered whole milk) was used in formula.
‡ Casein hydrolysate was used either in formula or intravenously.

excessive loss of nitrogen in the stool, which, decreased in a later period. Iron appeared to be well absorbed and utilized. Patients gained weight and showed increases in serum proteins and in hemoglobin during the periods of observation.

Fats tended to be poorly absorbed in the early postoperative period. The stools were found to contain an increased amount of fat, usually about 35 to 40% of stool solids being fats, as compared with normal values of 20 to 25%. The exact

3. Hawk, P. B., and Bergeim, O.: Practical Chemistry, Ed. 12, Philadelphia, The Blakiston Company, 1948.

4. Fortune, W. B., and Mellon, M. G.: Determination of Iron with *O*-Phenanthroline, Indust. & Engin. Chem., Anal. Ed. **10**:60-64, 1938.

mechanism involved in this failure of normal fat absorption was not at first apparent. It seems likely now that poor fat emulsification by bile is concerned. Possibly bile excretion is either deficient in amount or gall-bladder emptying is poorly correlated with feedings in the absence of mechanical or humoral factors involved when food from the stomach enters the upper small intestine. Certainly, it was noted that the addition of a commercial spreader tended to improve the rapidity of fat absorption. In other experiments the administration of bile along with the feeding seemed to have a similar result. Observations in one patient indicated that there was an ability to handle spontaneously fats administered in jejunal feedings, so that several weeks after jejunostomy repeated analyses for stool fats showed them to be present in decreasing amounts, approaching normal levels. We have noted similar variants in fat absorption following subtotal and total gastrectomy which add indirect evidence to the above conception of the difficulty encountered in fat absorption.

SUMMARY

Clinical and nutritional observations based on a study of 34 patients with the Maydl type of jejunostomy are presented. A proper technical procedure for performing this jejunostomy is included.

These studies demonstrate that an adequate nutritional intake can be pleasantly, safely, and conveniently obtained by this method of feeding.

GASTRIC AND JEJUNAL ALIMENTATION WITH FINE POLYETHYLENE TUBES

LAURENCE S. FALLIS, M.D.
AND
JAMES BARRON, M.D.
DETROIT

THE MAINTENANCE of nutrition is an ever-present problem in the management of both medical and surgical patients. The superiority of alimentary ingestion is well recognized, although emphasis in recent years has been directed toward the intravenous route. There are always some patients who cannot or will not take fluid and food by mouth. In this group until recently jejunostomy has been the only recourse when the usual type of gastric or jejunal tube has been poorly tolerated and the intravenous route has been unequal to the task of maintaining the patient in positive nitrogen, fluid, and electrolyte balance. The availability of fine caliber polyethylene tubes has changed this picture, since these small tubes are well tolerated in the upper gastrointestinal tract over a long time. Polyethylene tubes are available in various sizes.[1] We have found that size PE 200 with an outside diameter of 1.9 mm. is very satisfactory (Fig. 1). It is purchased in 100-ft. (3,048-cm.) lengths at a cost of about 12 cents per foot. For gastric use, the tubes are cut into 27-in. (69-cm.) lengths, but for jejunal use a 6-ft. (182.8-cm.) length is recommended. This extra length allows the proximal portion of the tube to be taped to the nose and the free end to be looped over the ear when not in use (Fig. 2).

When introducing gastric tubes in patients who can swallow, the tube is passed through the nares and into the stomach, as one would pass an ordinary stomach tube. Obviously a tube of this caliber is not rigid enough to be pushed into the stomach without the cooperation of the patient. For patients who cannot swallow, we have used rubber balloons weighted with 3 to 5 cc. of mercury, using plain absorbable surgical (gut) suture material to attach the tied end of the balloon to the end of the polyethylene tube (Fig. 3). This is readily accomplished by passing a 20 gauge needle through one side of the tube about 1.5 cm. from its distal extremity and out through the end of the lumen. A length of surgical gut is passed through the needle and tied after the needle is removed. Gastric juice disintegrates the surgical gut, allowing the balloon to pass on through the gastrointestinal tract.

The mercury-weighted balloon is introduced with the patient sitting up or with the head of the bed elevated from 45 to 60 degrees. The nasal passages are desen-

From the Department of Surgery, Henry Ford Hospital.

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

1. Polyethylene tubes are available from Clay Adams Co., Inc., 141 E. 25th St., New York 10.

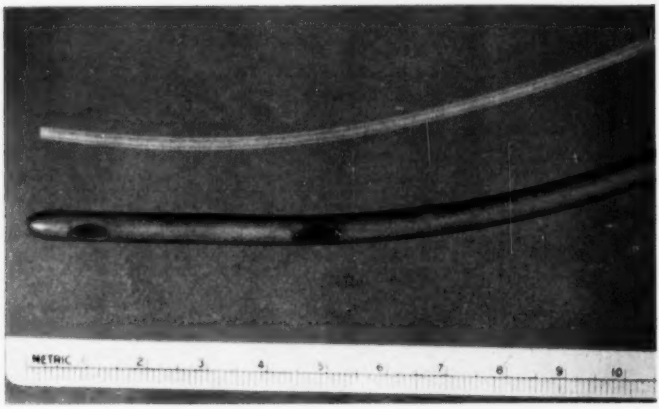

Fig. 1.—Ordinary rubber tube (below) compared to polyethylene tube size PE 200 (above).

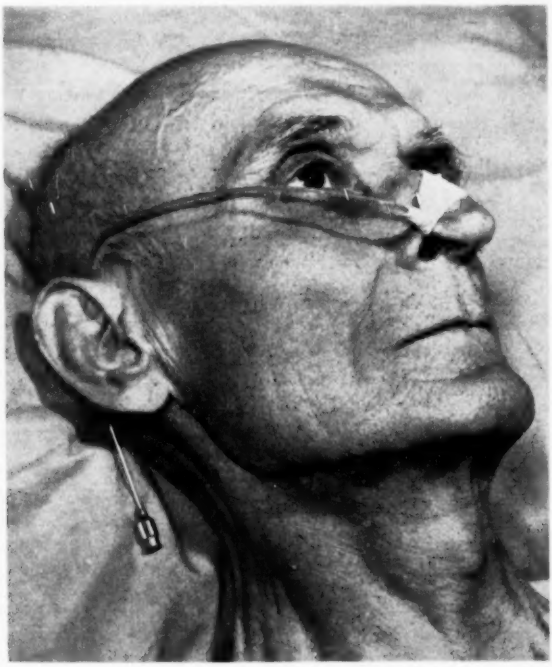

Fig. 2.—Polyethylene tube taped to nose. Free end is looped over ear when not in use.

sitized by applying 2% tetracaine (pontocaine®) hydrochloride in lubricating jelly, as suggested by Dr. J. M. Dondanville of our department of anesthesia. A cotton applicator is used to push the free end of the balloon well back into the nasopharynx (Fig. 4). The weight of the mercury will readily pull the tube down into the stomach even in a noncooperative patient. The tube is now taped to the nose and is ready for use. This method will be successful even in semiconscious patients.

In earlier trials we experimented with latex balloons of the same type used for Miller-Abbott tubes. However, these are semipermeable, and in certain instances the balloons became distended during their sojourn in the gastrointestinal tract. During the past year we have made use of ordinary "dime store" toy balloons measuring 1.5 to 2 cm. in width and from 7 cm. to 9 cm. in length. In addition to being cheaper, they show little tendency to absorb gases and become distended while in the gastrointestinal tract. We have also utilized the additional precaution, as

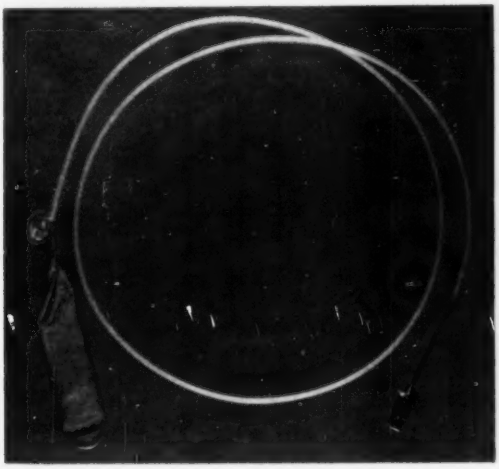

Fig. 3.—Gastric tube. Balloon is tied with nonabsorbable surgical (silk) suture material and then tied to end of tube with absorbable surgical (gut) suture material.

advocated by Cantor, of placing a small needle (No. 22) hole in the balloon near its base to serve as a safety valve (Fig. 5).

Polyethylene tubes 5½ to 6 ft. (165.1 to 182.8 cm.) in length have been placed in the jejunum when for various reasons the stomach could not be utilized for feeding. In this group there have been examples of gastric and duodenal fistulae, carcinoma of stomach, delayed emptying following gastrectomy, vomiting during pregnancy, etc. The bag is prepared as described previously. A needle hole is placed in the balloon to serve as a safety valve if necessary in case of balloon distention. Nonabsorbable surgical (silk) suture material (00 or 000) is then pulled through the section of polyethylene tube by means of a piano-wire stylet which is bent in the form of a hook on one end. When this wire is not available, the surgical silk may be passed by introducing it in the lumen of one end under water. Aspiration on the other end with a syringe will cause the surgical silk to be pulled through. The distal extremity of the string is tied to the end of the mercury-weighted balloon. A No. 16

needle is passed into the lumen of the proximal extremity of the tube to hold the surgical silk firmly in place (Fig. 6). Sufficient tension is put on the surgical silk thread to maintain approximation of the balloon to the distal end of the tube. Otherwise, if gastric suction is present, the string may be pulled up into the stomach tube. The balloon weighted with mercury is passed down into the stomach just as described

Fig. 4.—Cotton applicator is used to push mercury-weighted bag into nasopharynx.

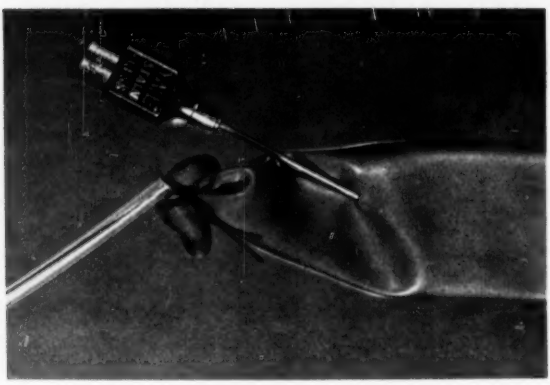

Fig. 5.—Needle (No. 22) is used to puncture balloon. Any air can be removed, and needle hole serves as safety valve in case of distention.

for the gastric tube. In general, it will be found that the fine polyethylene tubes will pass through the stomach and the duodenum much more readily than the usual larger gastrointestinal tubes. In the ordinary balloon for this type of tube 3 to 5 cc. of mercury are adequate, but when some edema or obstruction is present, extra mercury (up to 10 cc.) will prove helpful. The most difficulty in passing the tube will be

encountered in patients with little or no peristalsis, such as occurs in severe upper abdominal infections. By using as much as 8 to 10 cc. of mercury in the balloon and by utilizing various positions checked by fluoroscopy or x-rays and by ambulating the patient to the fullest extent, these tubes can be passed in nearly every case unless the upper gastrointestinal ileus is unusually severe. Often several days are required for passage of the tube. As soon as the tube passes into the jejunum for the desired distance it is taped to the nose and the free end looped over one ear when not in use. The needle is removed and the string is pulled through the tube and intestinal tract by peristaltic action of the balloon. In every case one should make sure either by questioning or by x-ray examination that the balloon and string have been passed. Surgical gut cannot be used to attach the balloon to the tube in jejunal tubes because the gut will dissolve too quickly if there is any delay in passage through the stomach or the duodenum. Care must be taken not to kink these tubes because partial blockage

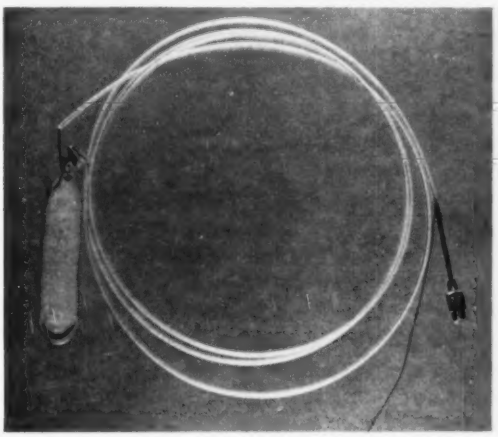

Fig. 6.—Jejunal tube 5½ to 6 ft. (165.1 to 182.8 cm.) in length. Nonabsorbable surgical (silk) suture material is passed through lumen to hold mercury-weighted balloon.

will occur. The string and balloon should not be released in any patient who has obstruction in the colon or lower small intestine.

These fine polyethylene tubes have been used for replacing various body fluids, such as gastric juice, bile, pancreatic secretions, duodenal secretions, and upper small bowel contents. Water, water-soluble drugs, and feeding formula are readily passed by this tube. These tubes are well tolerated for prolonged periods. In children with nephrosis no evidence of irritation was evident after several months of continuous use.

After many trials, the following high caloric feeding formula which will readily drip through this small polyethylene tube has been adopted:

Homogenized milk	500 cc.
Ledinac® (a preparation of liver protein hydrolysate, thiamine, and folic acid) or "Stuart amino acids" (a casein hydrolysate preparation)	175 gm.
Cerelose® (a form of glucose)	300 gm.
Eggs (whole)	4
Klim® (a brand of powdered whole milk)	75 gm.

In preparing the feeding formula, the following steps are used: The cerelose* is dissolved in a portion of the milk which has been warmed. This is then set aside to cool. The klim* is then strained through a wire sieve to remove any lumps that may be present. This is then added to the remainder of the cool milk. This is stirred at once to prevent any lumps. The eggs and ledinac* (or "Stuart amino acids") are then added to this cool solution. Finally the cerelose* and milk which have cooled are then mixed with the above. The whole preparation is strained through cheesecloth and then placed in a refrigerator until used. With reasonable care and experience in preparation there will be little tendency for clogging of the tube. The formula per quart contains 146 gm. of protein, 71 gm. of fat, and 423 gm. of carbohydrate. The caloric content is 2,914 cal. per quart, or approximately 3 cal. per cubic centimeter. The material cost is less than $1.50 per quart when ledinac* is used. Ledinac* and "Stuart amino acid" preparations have been chosen because they are water soluble. Ledinac* is somewhat cheaper than the other, but the solution will be slightly thicker. Water-soluble vitamins and drugs must be used, otherwise the small tube will become plugged. Since we have used only simple drip apparatus for feeding into the tube, poly-vi-sol* (a water-soluble liquid multivitamin preparation) has been used as the main source of vitamins in our feedings.

The various preparations are administered through the polyethylene tube by means of intravenous-injection bottles with the attached drip and plastic intravenous-injection tube (Fig. 7). We have not used the mechanical pump described by Snyder [2] but realize its value in certain instances when there is a shortage of help.

In general we have found that one must start gastric or jejunal feeding and especially the latter with minimal amounts of liquid or formula, not more than 20 cc. or 30 cc. at a time. We have learned to gradually increase the feeding daily until the desired level is reached. In many patients we have started with these small amounts every one to two hours and have worked this up gradually to as much as 200 to 300 cc. every hour. Diarrhea, cramps, nausea, and vomiting are the main disagreeable features of tube feeding. Camphorated opium tincture (2 fluidrachms [8 cc.] four times a day) and methantheline (banthine*) bromide (up to 100 mg. four times a day), have been used liberally until the patient is well adjusted to the feedings. Each of these medicines can be easily added to the feedings. Additional experience has shown that small, gradually increased feedings have done a great deal to reduce the incidence of cramps and diarrhea. The results will vary directly with the interest and patience that is applied in these cases.

This type of polyethylene tube has now been used in well over 150 cases in the past several years. These have included duodenal fistula (two cases), gastrocolic fistula (two cases), pancreatic fistula (one case), delayed emptying following gastrectomy (seven cases), oral surgery (cleft palate, carcinoma of tongue, lip, maxilla), carcinoma of pharynx and esophagus, nephrosis and nephritis, swallowing problems, cerebrovascular accidents, brain tumors, anorexia, biliary fistula, high small intestinal fistula, coma, partial upper gastrointestinal obstruction, and vomiting during pregnancy.

The jejunal tube has been of great value in two patients with gastrocolic fistula. The tube was easily passed in these two patients. In the first patient healing was

2. Snyder, C. C.: The Continuous Forced Feeding Pump: A New Approach to Nutritional Therapy, Plast. & Reconstruct. Surg. **8**:73-75 (July) 1951.

spontaneous. The second patient, who had severe cardiac disease, improved greatly and was successfully operated on (Fig. 8). The tube has been used with excellent results in seven cases of delayed emptying following gastrectomy. In these we have been able to replace fully all fluid aspirated from the stomach and to add feeding

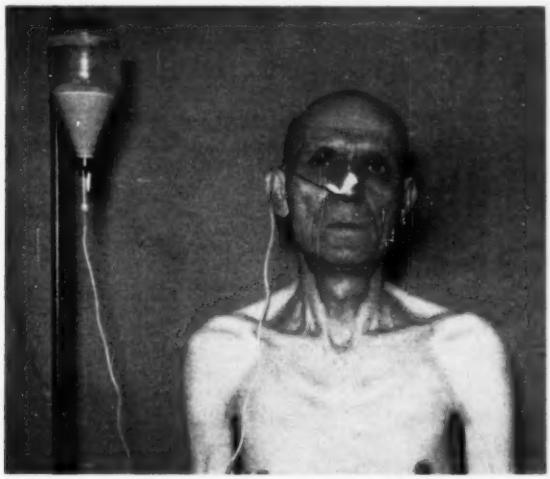

Fig. 7.—Liquids placed in intravenous-injection bottle with drip and plastic tube attached.

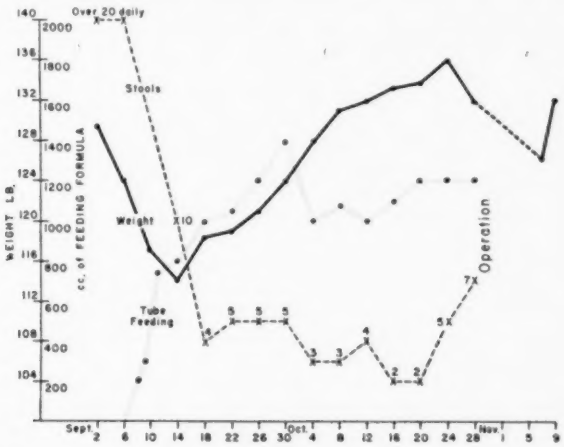

Fig. 8.—Gastrojejunocolic fistula progress in Patient R. B., aged 62. Stools decrease and weight increases as tube feedings are started through jejunal tube. Tube was inadvertently pulled back into stomach shortly before surgery, causing increase in number of stools and slight weight loss.

formula plus water and medicines as desired. In addition to greatly easing the electrolyte and nutritional problem, the freedom from the intravenous use of needles has been greatly appreciated by the patients. In most patients we have been able to stop the weight loss and in many instances actually produce a gain in weight.

SUMMARY AND CONCLUSIONS

Small polyethylene tubes are well tolerated for long periods when used for gastric or jejunal intubation.

A technique for passing these tubes into the stomach and the jejunum has been described.

In the majority of cases these simple inexpensive tubes will make jejunostomy unnecessary.

A simple inexpensive feeding formula has been presented.

Since presenting the material in this paper, the authors have been using liquefied and homogenized whole food for tube feedings and have found that it is tolerated better than any feeding formula previously employed.

DISCUSSION

DR. HENRY RANSOM, Ann Arbor, Mich.: For several years we have been using the formulas for jejunostomy feeding which were developed by Dr. Zollinger and his associates, and the older empirical methods, which were almost always unsatisfactory, have been replaced by this much more efficient and rational method.

In reviewing the records of patients with carcinoma of the stomach seen in our clinic, we have noted that during recent years, in nonresectible lesions, exploratory laparotomy only has been performed far less frequently than in years gone by, and in its stead, laparotomy with jejunostomy has often been performed. This probably indicates the opinion of many of our surgeons regarding the value of a feeding jejunostomy for terminal care. However, I believe that we often delude ourselves regarding the true value of such expedients. A few years ago Drs. Donald Cooper and Robert Buxton reviewed the results of all cases of gastrostomy performed in our hospital. In cases of nonremovable cancer of the stomach or esophagus, it seems problematical whether such a method for artificial feeding is really worth while. The matter, of course, is an individual one, and the surgeon often feels compelled to make some effort at palliation during the last days of life for the sake of the family and for the psychological effect on the patient.

With regard to jejunostomy during the period of preparation for surgery or as a complementary procedure at the time of operation, we are dealing with a different problem. The group of patients needing extensive preparation for operation has been well discussed by Dr. Boles. I think we have been somewhat less enthusiastic about this method than we might be, but we now try to remove the offending lesion as soon as possible, first getting the patient into the best possible condition by multiple transfusions and the judicious use of intravenous fluids along with other auxiliary measures, rather than by jejunostomy.

A complementary jejunostomy often is of great value. One of the best subjects is the patient who has had a total gastrectomy. Since these patients often have a considerable amount of difficulty in taking an adequate amount of food for some time after operation, if jejunostomy feedings can be added to that which is taken by mouth, some of the nutritional problems are solved during the trying convalescent period. It was noted by Drs. Cooper and Buxton in their gastrostomy series that when patients can be given some food by mouth in addition to gastrostomy feedings, the nutritional improvement is greater than when all the caloric intake is by jejunostomy or gastrostomy alone. In the postoperative period when some complication has arisen, jejunostomy frequently is useful. For example, with a leaking esophagogastric anastomosis, a blow-out of the duodenal stump, or a malfunctioning gastrojejunostomy after subtotal gastrectomy, jejunostomy will permit feeding an adequate diet while the diseased area is put at rest, thus allowing the inflammatory process to subside. These are always difficult postoperative complications to deal with, but with the method which was described this morning, in many cases the patient can be supported for 10, 14, or 21 days, or even longer, and lives are thus saved.

DR. SAMUEL A. SWENSON, JR., Omaha, Neb.: I should like to discuss Drs. Brintnall, Daum, and Womack's paper.

As you all know, the Maydl jejunostomy was originally put forth as an experimental procedure to fistulize the gastrointestinal tract at the jejunum. I used it in the case of anorexia nervosa in which a little girl's condition had reached such a point that she could take nothing by mouth and could keep nothing down.

We attempted tube feedings, and she had vomited feedings around the tube. Her condition became progressively worse until she could take nothing at all by mouth, and she looked like someone who had come from a concentration camp.

We did a Maydl jejunostomy with the use of anesthesia in a hospital in a small town and continued her feedings through the jejunostomy. That was two years ago. She is now playing in the marching band in high school and has gained weight and has had normal development since that time.

The Maydl jejunostomy is a procedure that will allow alimentation below the ligament of Treitz where there is no possibility of regurgitation into the stomach. The distal loop of the jejunum is used; this has a natural peristaltic action carrying the feedings on down into the gastrointestinal tract without regurgitation if the distal loop is long enough.

I certainly have been well pleased with the results in this case.

DR. ROBERT M. ZOLLINGER, Columbus, Ohio: As Dr. Boles has said, we find that jejunostomy may occasionally be worth while, especially in the elderly poor-risk patient who has lost a great deal of weight. Although we have not utilized the Maydl type jejunostomy described in Dr. Brintnall's paper, I think it has much to offer. We would be inclined to use this technique in terminal cases, because I am sure the patient would be much more comfortable than if he had a tube gastrostomy or a tube jejunostomy (Stamm) for a prolonged period.

I think that the tube-feeding method described by Dr. Brintnall is very valuable. [Slide] We have utilized jejunostomy for several years and have found it very useful in the type of case Dr. Brintnall described. It is especially useful in the treatment of burns in children. A continuous flow of nutrient is achieved by employing a force pump which is equipped with a thermos bottle for keeping the temperature of the solution constant. The flow can be regulated so that the patient is fed steadily throughout the 24-hour period.

[Slide] This is the weight curve of a patient with chronic calcareous pancreatitis. You will observe on the left that the patient was taking only 600 cal. a day because every time he ate he had considerable distress. However, by means of the method described by Dr. Brintnall, utilizing the force pump and continuous feeding, the calories were increased gradually so that the patient's intake approached 4,000 cal. a day. He gained 10 lb. (4.5 kg.) in two weeks.

I believe it is important to utilize a variety of methods for improving or sustaining the nutrition in patients unable to take enough food by mouth. Improved nutrition lowers the morbidity and mortality in poor-risk depleted surgical patients.

CLINICAL STUDIES WITH THE ELECTROGASTROGRAPH

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AND
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THE EXISTENCE of an electrical potential difference across the gastric wall has been known for over a hundred years. Crane and co-workers [1] refer to the work of Donné in 1834 as probably the earliest measurement of this potential difference. In 1865 Rosenthal [2] while investigating the potential difference across the skin of a frog also measured it across the mucosae of the stomach and intestines. Since then numerous attempts have been made to measure and evaluate clinically the gastric potential differences, but the work has progressed slowly because of technical difficulties and the complex nature of the gastric function.

Investigation has proceeded along two basic lines, some investigators attempting to measure only the steady D. C. component, while others have measured only the small superimposed A. C. components. A few investigators have followed a middle course, trying to show the over-all pattern, but owing to the limitations of the equipment used their records have not clearly presented the information.

A brief review of the important papers in the field is as follows:

In 1922 Alvarez and Mahoney [3] described action currents obtained from various points of the gastrointestinal tract. They worked with anesthetized animals and applied the electrodes to the serosal surface, measuring only the A. C. components. Later that year Alvarez [4] made the first human electrogastrographic recording. In 1924 Richter [5] showed records of action currents from the stomach which were remarkably similar in wave form to those produced by the electrocardiograph, except that the gastric-wave complexes were of longer duration and of slower frequency and were associated with gastric contractions. Puestow [6] also recorded simultaneous electrical and mechanical changes and obtained results similar to those of Richter. In 1933 Berkson [7] showed that although the electrical changes

Supported by a grant from the National Cancer Institute of Canada.

Facilities of the Clinical Research Unit, Queen Mary Veterans Hospital, were used.

From the Sherbrooke Hospital (Dr. Martin), and the Royal Victoria Hospital (Mr. Morton).

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

1. Crane, E. E.; Davies, R. E., and Longmuir, N. M.: Biochem. J. **43**:321-336, 1948.
2. Rosenthal, I.: Arch. Anat. u. Physiol. **32**:301, 1865.
3. Alvarez, W. C., and Mahoney, L. J.: Am. J. Physiol. **58**:476, 1922.
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5. Richter, C. P.: Am. J. Physiol. **67**:612, 1924.
6. (*a*) Puestow, C. G.: Proc. Soc. Exper. Biol. & Med. **29**:901, 1932; (*b*) Arch. Surg. **24**:565, 1932.
7. (*a*) Berkson, J.; Baldes, E. J., and Alvarez, W. C.: Am. J. Physiol. **102**:683, 1932. (*b*) Berkson, J.: ibid. **104**:62, 1933.

and muscular contractions usually were synchronous, by the use of appropriate drugs one record could be abolished while the other continued, and vice versa. Recently (1950) in the clinical field Schuster and Schmucker [8] have examined the changes in the activity of the electrogastrogram in response to a test meal. They have also observed that in cases of peptic ulcer or hyperacidity there are characteristic changes in the electrical activity. Fukushima and co-workers [9] have also shown changes in wave form associated with peptic ulcer and gastric spasm, and they state that they can diagnose gastric carcinoma on the basis of specific patterns.

Concerning the D. C. component of the gastric potential difference, much investigation has been done by Rehm.[10] Working with gastric mucosa from dogs, he has tried to associate the magnitude of electrical energy available from that mucosa with the energy required for the secretion of HCl. He has also investigated the change of D. C. potential in response to stimulation. Crane and others [1] have carried out somewhat similar studies on the gastric mucosa of frogs. Rice and Ross [11] have made electrogastrographic studies on patients and on dogs and have concentrated their interest on the change of D. C. level in response to the administration of drugs and to drinking milk, etc. They have outlined some of the technical problems in obtaining the recordings.

Following the middle course and concerning themselves with clinical application, other authors [12] have studied the change in D. C. level in response to the swallowing of fluids or to the injection of drugs. They noted that there were gross irregularities superimposed on the D. C. level, and on the basis of these irregularities, the D. C. level and the response to fluids, they have made clinical diagnoses. Their records, however, did not show any regular rhythmical activity, such as was shown by Schuster and Schmucker [8] or Fukushima and co-workers,[9] because the amplitude of this activity was too small to be recorded by their apparatus. A recent article by Goodman and others [13] describes improvements made in their instruments and indicates that clinical records showing this rhythmical activity, together with the D. C. level, have been made and are yet to be published.

An outline of some of the technical problems involved in obtaining the electrogastrographic recording is given by Ravin and co-workers.[14]

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9. Fukushima, K.; Okada, I., and Nakasono, Y.: M. J. Osaka Univ. **2**:83, 1951.

10. (*a*) Rehm, W. S.: Am. J. Physiol. **139**:1, 1943; (b) ibid. **140**:720, 1944; (*c*) ibid. **141**:537, 1944; (*d*) ibid. **144**:115, 1945; (*e*) ibid. **147**:69, 1946. (*f*) Rehm, W. S., and Hokin, L. E.: ibid. **149**:162, 1947; (*g*) ibid. **154**:148. (*h*) Rehm, W. S., and Enelow, A. J.: Gastroenterology **3**:306, 1944. (*i*) Rehm, W. S.: ibid. **14**:401, 1950. (*j*) Rehm, W. S.; Hokin, L. E.; de Graffenreid, T. P., II.; Bajandas, F. J., and Coy, F. E., Jr.: Am. J. Physiol. **149**:77, 1947.

11. Rice, H. V., and Ross, R. T.: Am. J. Physiol. **149**:77, 1947.

12. (*a*) Adair, G. S., and Goodman, E. N.: J. Physiol. **87**:35, 1936. (*b*) Goodman, E. N.: Surg., Gynec. & Obst. **75**:583, 1942. (*c*) Quigley, J. P.; Barcroft, J.; Adair, G. S., and Goodman, E. N.: Am. J. Physiol. **119**:763, 1937. (*d*) Sawyer, P. N.; Rhoads, J. E., and Panzer, R.: Surgery **26**:479, 1949.

13. Goodman, E. N.; Ginsberg, I. A., and Robinson, M. A.: Science **113**:682, 1951.

14. Ravin, I. S.; Kneisel, J. J., Jr.; Taylor, R.; Lemon, H. M.; Thompson, E. M., and Smithwick, R. H.: Measurement of Electropotentials of the Stomach, in Surgical Forum: Proceedings of the Forum Sessions, Thirty-Sixth Clinical Congress of the American College of Surgeons, Boston, October, 1950, Philadelphia, W. B. Saunders Company, 1951, p. 69.

TECHNIQUE

The most important problem in obtaining the recordings is the electrodes. The existence of polarization, contact potentials, and liquid junction potentials must be recognized, and methods must be used which compensate for, or eliminate, these factors. Most of the earlier workers used silver-silver chloride electrodes to eliminate polarization, and the contact potential was balanced out because only the A. C. components were being examined.[15] Other workers who attempted to eliminate contact potentials have used calomel electrodes connected to the tissues under study by tubes containing a solution of KCl or HCl.[16] These electrodes are highly reproducible, but they are cumbersome, and a serious drawback to their use is the tendency of air bubbles to form in the connecting tubes.

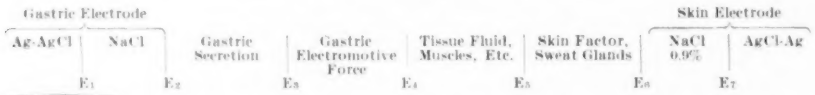
The present studies have been made using a silver-silver chloride electrode similar in principle to that described by Burr [17] and Beutner.[18] It consists of Ag-AgCl in contact with isotonic sodium chloride solution, the electrical contact with the tissues being made by cotton wicks soaked in saline. The construction of the electrode is similar to that described by Rice [19] but with several modifications. A long flexible plastic tube *2* mm. in diameter encloses the wire and terminal electrodes, and projects *3* to 5 cm. beyond the AgCl tip. This terminal piece of plastic is filled with saline and then plugged with a cotton wick. The electrodes are reproducible to within 1 or *2* mv., and once made they must be treated with some care, since they are photosensitive. If two similar electrodes are used, the metal-liquid contact potential is neutralized. Liquid junction potentials, however, are created with this electrode: For example, if the stomach is actively secreting HCl and the gastric juice is approximately pH 1, then there will be a diffusion or liquid junction potential of *20* to 30 mv. between the saline of the electrode and the HCl of the gastric juice. The second electrode is placed in contact with the outer aspect of the arm; here the sweat glands are minimal, and therefore artefacts due to change of skin potential are reduced.[20] Liquid junction potentials need not be considered here, since the two fluids in contact have very similar compositions.

The resultant potential difference between the two electrodes is amplified by a vacuum-tube D. C. amplifier. The gain is sufficient to give a sensitivity of *2* mv. per 1.3 cm. deflection of the recording pen. There is a provision in the amplifier for the balancing out and measuring of the D. C. potential so that maximum sensitivity of *20* mv. per full scale deflection may be used at all times, regardless of the D. C. level.

The recorder is a 0 to 1 ma. Esterline-Angus recording milliammeter. This meter has a response time sufficiently great that some of the high frequency transients in the electrogastrogram are recorded but not so high a response that there is interference from the electrocardiogram. The response time varies with damping and paper speed, but with our equipment a 90% response occurs in approximately 0.5 seconds. It has a paper-chart drive of variable speeds from 1.9 cm. an hour to 30 cm. a minute in 10 steps. The recording speed used in this work is 1.9 cm. a minute. The chief disadvantage of this recorder is the curved coordinates, but by operating at the center of the chart this is minimized.

FACTORS CONTRIBUTING TO THE POTENTIAL DIFFERENCE MEASURED

The following factors affect the resultant potential difference between the skin and the gastric electrodes and may be represented diagrammatically as follows:



15. (*a*) Johnson, C. A., and Laing, G. H.: Am. Heart J. **20**:160, 1940. (*b*) Tumpeer, I. H.: Am. J. M. Sc. **184**:831, 1932; (*c*) Ann. Int. Med. **10**:487, 1936. (*d*) Alvarez.[3] (*e*) Richter.[5] (*f*) Puestow.[6a] (*g*) Berkson.[7a]

16. Footnotes 10a, 12a-d, and 14.

17. Burr, H. S.: M. Physics, pp. 1117-1121, 1944.

18. Beutner, R.: M. Physics, pp. 35-38, 1944.

19. Rice, H. V.: Personal communication to the authors.

20. Rice, H. V.: Read before the Royal College of Physicians and Surgeons Convention, Quebec, Canada, September, 1951.

The vertical lines represent phase boundaries at which there may exist a potential difference. E_1 and E_7 are integral potentials of the electrodes and neutralize each other if the electrodes are identical.

E_2 represents the diffusion potential difference between the gastric secretion and the N/6 NaCl of the gastric electrode. The magnitude of this potential difference varies with the logarithm of the ratio of the ionic mobilities and concentrations of the two solutions in contact. In this particular case the most important factor is the hydrogen ion of HCl, owing to its high mobility and relatively high concentration. The more concentrated the HCl or the more dilute the NaCl in the electrode, the greater will be this liquid junction potential. With the use of data from Glasstone,[21] the potential difference for N/6 NaCl and N/10 HCl is calculated to be 19 mv., and this agrees very well with the value of 22 mv. measured by Hokin and Rehm.[22] The polarity is such that the NaCl solution is positive with respect to the HCl.

The gastric electromotive force has been localized in the dog by Rehm [10e] to the region between the surface of the mucosa and the submucosa. The mucosa is negative with respect to the submucosa, and in the case of the nonsecreting stomach the potential difference has a magnitude of approximately 80 mv.

The contribution of body tissues, fluids, skin factor, etc., has not been evaluated, although Rice [20] points out that the polarity of the potential difference across the sweat glands opposes that of the gastric potential difference. As observed earlier, it is felt that the liquid junction potential between sweat and the NaCl of the electrode may be neglected.

The electrogastrogram is then a function of the following variables:

A. Those affecting the D. C. or steady level
   1. Electrode position on the gastric mucosa
   2. Gastric tone
   3. Metabolic activity of mucosal cells
      (*a*) Gastric electromotive force
      (*b*) Liquid junction potential with HCl
   4. Skin factors

B. Those affecting the A. C. or changing potential difference
   1. Neuromuscular activity
      (*a*) Peristalsis
      (*b*) Changes of tonus
   2. Mechanical characteristics of the electrode

Rosenthal,[2] Rice and Ross,[11] and Crane and others [1] have shown that the gastric potential difference is greatest in the region of the fundus and becomes less toward the pylorus so that the potential difference measured will vary with the electrode position. That tone is probably a contributing factor to the D. C. level will be pointed out in a later section dealing with the response to swallowing milk.

21. Glasstone, S.: The Electrochemistry of Solutions. London, Methuen & Co., Ltd., 1930; Textbook of Physical Chemistry, Ed. 3, New York, D. Van Nostrand Company, Inc., 1949.

22. Hokin, L. E., and Rehm, W. S.: Am. J. Physiol. **151**:380, 1947.

The metabolic activity of the mucosal cells appears to be responsible for the generation of the gastric electromotive force, for Coy and co-workers,[23] Rice and Ross,[11] and Crane and others [1] have observed that the potential difference rapidly falls if the blood supply to the mucosa is interrupted. Thus conditions which reduce the metabolism of the mucosal cells would be expected to cause a reduction of the gastric potential difference (i. e., it would be less negative). It should be noted that increased secretion of HCl causes an increase of the diffusion potential difference with the NaCl solution of the electrode in such a direction that the gastric electrode becomes less negative.

The various components of gastric contractions show up in the wave forms of the electrogastrogram, but these are undoubtedly a function, to some degree, of the mechanical characteristics of the electrode. A large moderately stiff electrode placed a few inches within the stomach will change its contact with every gastric contraction, and it may also stimulate abnormal contractions. A fine flexible electrode coiled in the stomach and held in contact with the mucosa by the light spring-like action of the tubing appears to cause little artefact in recording normal gastric motility. However, when there is hypermotility this electrode certainly is moved about in a manner dependent on its elasticity and flexibility, and it may therefore be expected to contribute to some of the sudden changes in the wave forms seen in these recordings.

CLINICAL STUDIES

This study is based on 100 recordings made on 60 patients. The recordings were usually made in the morning, two to four hours after breakfast, or sometimes in the afternoon, one to three hours after lunch. The fasting stomach was not considered essential, since the objective was to see just what could be recorded from the stomach and the responses to stimuli. The electrode was inserted into the stomach, via the nose, to a depth of 33 in. (83.8 cm.). A roentgenological check of the position was done in a number of cases before and after recording, and the electrode tip was usually found to be in contact with the fundus or with the greater curvature; occasionally the electrode passed into the duodenum. Figure 1 shows a typical position of the electrode before and after recording.

In previous papers there has been considerable variation in the convention of signs of the recordings, and this leads to confusion in the interpretation of the various responses. Since it is the gastric potential difference in which we are interested, we should plot it as the dependent variable and specify its potential difference with respect to the skin, which is considered arbitrarily to be zero. The usual mathematical convention with regard to + and — is adopted: An upward deflection represents a change in the positive direction and a downward deflection represents a change in the negative direction. Each large vertical division represents 2 mv., and the time scale is one minute per division.

A. Normal Electrogastrogram.—Rhythmical electrical changes have been demonstrated by numerous investigators.[24] Alvarez [25] has shown waves at the rate of

23. Coy, F. E., Jr.; Banjandas, F. J.; de Graffenreid, T. P., II, and Rehm, W. S.: Gastroenterology **17**:260, 1951. Rehm.[10c]

24. Footnotes 8 and 15a-g.

25. (*a*) Alvarez, W. C.: An Introduction to Gastro-Enterology (Third Edition of the Mechanics of the Digestive Tract), New York, Paul B. Hoeber, Inc., 1939; (*b*) Alvarez.[3, 4]

3 cycles per minute (cpm) and very similar to those in the records obtained mechanically. He has also demonstrated components of 9 to 11 cpm. The wave forms obtained by Schuster and Schmucker [8] and by Fukushima and his co-workers [9] are very similar to ours.

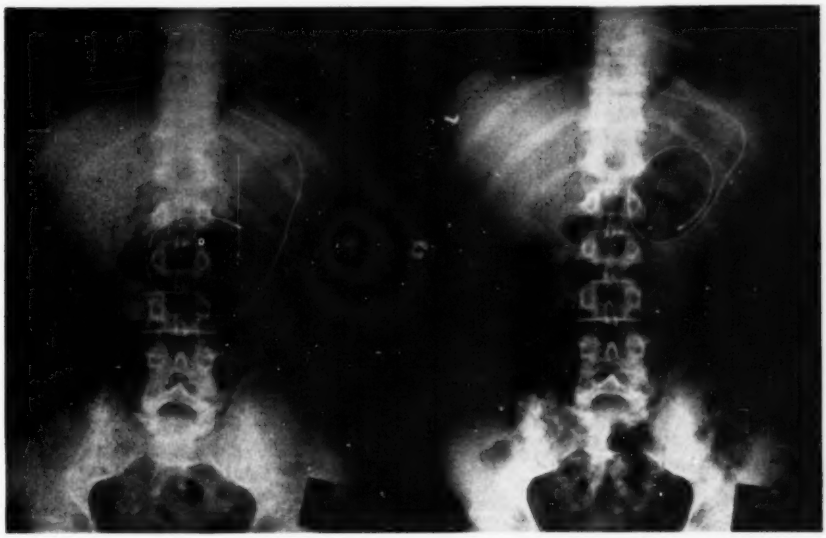


Fig. 1.—Electrode position before and after recording.

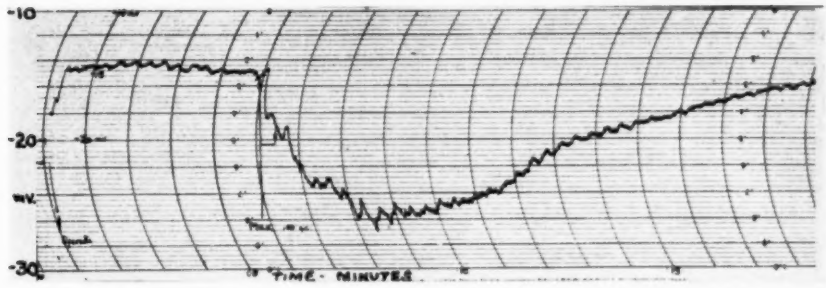


Fig. 2.—Normal electrogastrogram showing 3 cycles per minute activity and typical response to drinking milk. D. C. level is —15 mv.

Figure 2 shows a typical recording. The D. C. level is — 15 mv., 3 cpm activity of 1 mv. amplitude is present, and a response to milk of — 10 mv. is observed.

This response has caused much controversy. Some authors assert that it is positive; others, negative, and still others state that it may be either.[26] In our recordings the response to drinking fluids has had a latency of 0 to 60 seconds, an

26. Footnotes 10h and 12a-d.

increased negativity of 5 to 15 mv., and a duration of 3 to 10 minutes. On only four occasions was the response positive; this was probably due to diffusion potentials.

Rehm [10b] concludes that Goodman's [12b] observations are unacceptable, since the negative response is explainable by diffusion potentials. This may be true for electrodes filled with N/10 HCl as used by these investigators, but for electrodes using NaCl solution as contact, the change in diffusion potential when milk dilutes the acid surrounding the electrode would be expected in the positive direction. This was confirmed experimentally, with use of the apparatus shown in Figure 3.

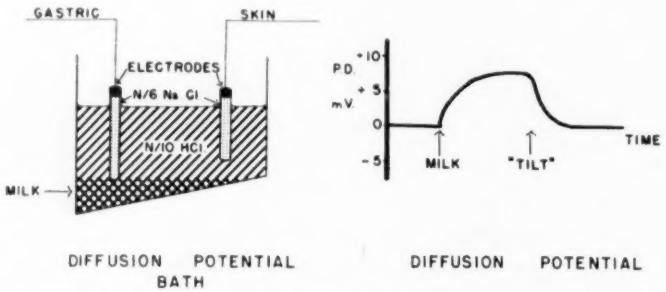


Fig. 3.—Change of diffusion potential caused by addition of milk to N/10 HCl surrounding "gastric" electrode.

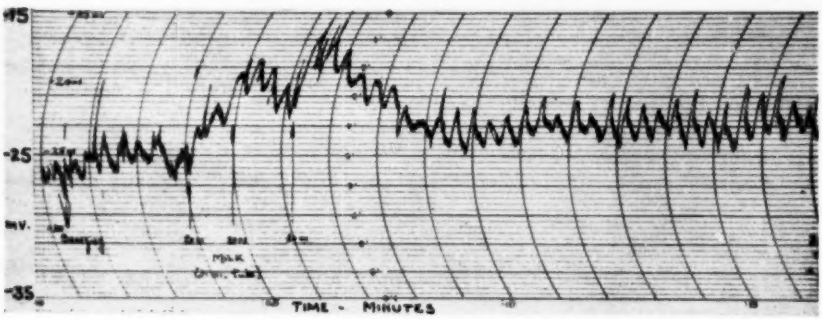


Fig. 4.—Positive response caused by injecting milk down second lumen of special gastric electrode.

The electrodes were immersed in a bath of N/10 HCl, and, since the system balanced, there was zero potential difference between the electrodes. Milk was then slowly added to the solution surrounding the "gastric electrode," and this electrode became positive. The bath was then tilted so that the milk could diffuse around the "skin electrode" as well, and the potential difference returned to zero.

A special gastric electrode had been made with concentric tubes for simultaneous recording and the aspiration of stomach contents. This made it possible to inject milk into the stomach around the electrode. In this case the change of potential was positive, corresponding with the results expected if the response was due to diffusion potentials. An example of this response is shown in Figure 4.

The explanation of these positive or negative responses seems to be as follows: The small electrode used in these recordings usually lies in contact with the greater curvature, but fluid usually passes into the stomach along the magenstrasse [27] and therefore does not come in contact with the electrode. It is only when the electrode is on the lesser curvature that the fluid surrounds it, thus causing a change of diffusion potential and a positive response. Cannon and Leib [28] have observed that there is a tonic relaxation of the stomach on swallowing, and it is probably this change of tonus which causes the negative response. It is possible that the electrode changes its position with this relaxation, but the responses are so similar each time that this does not seem likely.

Recordings were made in 14 normal patients, and the D. C. component ranged from — 35 to + 5 mv., with an average mean of — 14 mv. This is lower than the values recorded by other observers and is probably due to the different electrodes used and the varying stages of gastric activity.

Frequency components higher than 3 cpm have been noted on some recordings, but it has been difficult to distinguish these from possible respiratory artefacts.

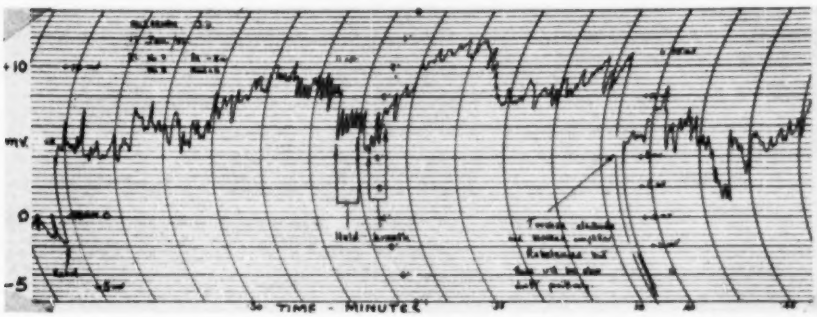


Fig. 5.—Presence of 11 cycles per minute activity while patient was holding his breath.

That these frequencies do exist in certain cases is shown in Figure 5 in which waves of 11 cpm continued while the patient held his breath—this patient had increased gastric irritability.

B. Effect of Electrode.—The size of the electrode may alter the normal gastric motility. When a less flexible electrode of double the usual diameter was used, the electrogastrographic recording of a certain patient was interpreted as being positive and exhibiting slow waves of period 3 to 5 minutes and amplitude 5 to 10 mv. Shortly afterwards the patient complained of nausea, and when the electrode was withdrawn 4 or 5 in. (10.1 to 12.7 cm.) this feeling passed; the gastric potential difference became negative, but the slow waves of large amplitude continued. Later, when the usual more flexible electrode was used the electrogastrographic recording was interpreted as normal for this patient. These waves are demonstrable in other recordings, and a good example is shown in Figure 7*C*.

27. Babkin, P. B.: Secretory Mechanism of the Digestive Glands, New York, Paul B Hoeber, Inc., 1944; Alvarez.[25a]

28. Cannon, W. B., and Leib, C. W.: Am. J. Physiol. **29**:267, 1912.

C. Peptic Ulcer.—The recordings of patients with peptic ulcer have indicated characteristic wave forms, and in this respect our recordings resemble those of Schuster and Schmucker [8] and Fukushima and co-workers.[9] Figure 6 illustrates a typical recording. This patient had had a hemorrhage from a duodenal ulcer,

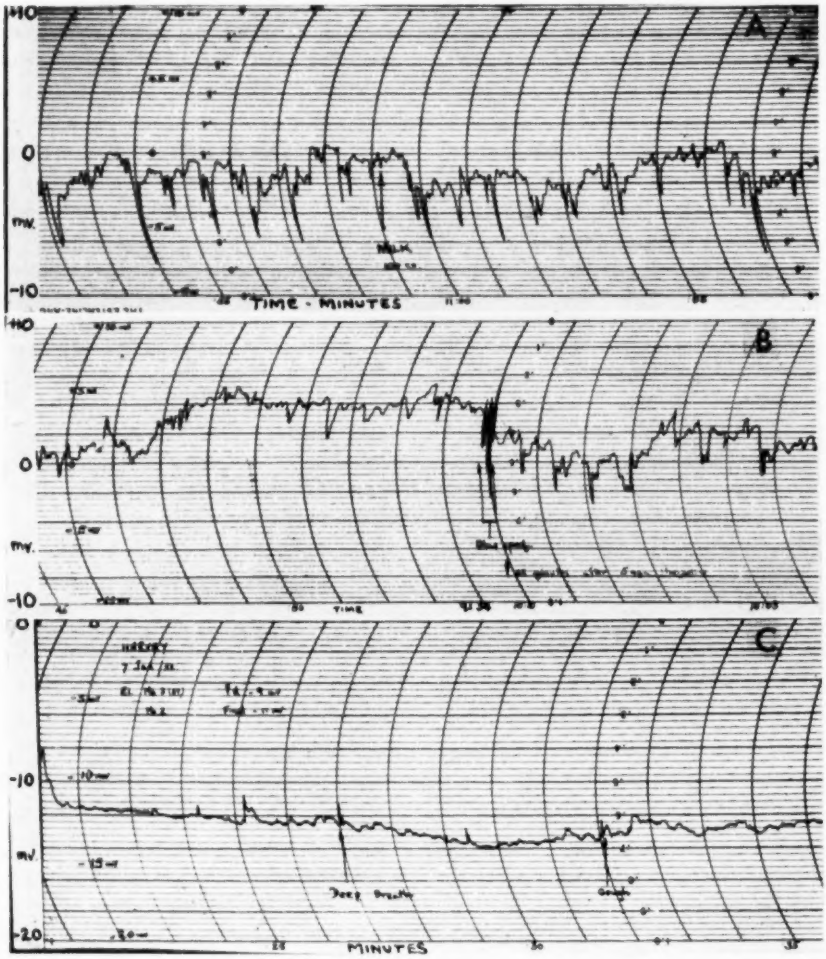


Fig. 6.—Response to duodenal ulcer (hemorrhage), (*A*) before treatment; (*B*) after treatment with methantheline (banthine*) bromide for six days; (*C*) after treatment with methantheline bromide for three weeks.

and Figure 6*A* is the recording obtained before therapy was started. The average D. C. level was — 2 mv., and there were irregular spikes of 2 to 4 mv. He was given 50 mg. of methantheline (banthine*) bromide four times a day, and after six days the recording shown in Figure 6*B* was obtained. The average D. C. level

then was + 2 mv., but the wave form was not so irregular as before; the second half of this record shows wave forms similar to those in the initial recording and were obtained as a result of administering 5 mg. of bethanecol (urecholine®) chloride three-quarters of an hour before the recording was made. After therapy

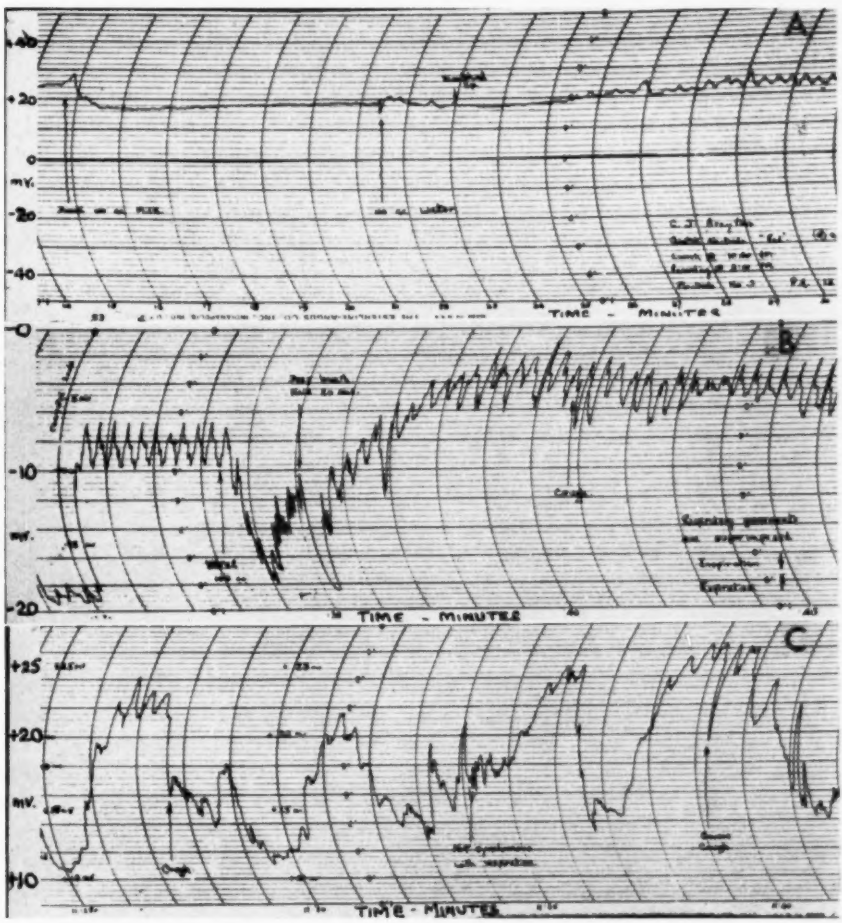


Fig. 7.—*A*, recording made July, 1951, of presumably normal stomach. D. C. level is 2— mv. *B*, recording one week after gastric hemorrhage. Patient was given antispasmodics and was clinically improved. *C*, no medicines were given for three days following treatment described above (*B*). Patient felt nauseated.

was given for three weeks, the D. C. level was — 12 mv., and low amplitude activity was seen (Fig. 6*C*).

The next patient was most interesting, since he had been recorded in July, 1951, as a normal subject. The average D. C. level then was + 20 mv., and this confused

the interpretation of the original recordings. This is shown in Figure 7*A*. The recording was made at a sensitivity of 10 mv. per division. Five months later he was readmitted with pneumonia. He then had a gastric hemorrhage, and investigation disclosed gastritis, a small duodenal ulcer, esophageal varices, and cirrhosis of the liver. In spite of this two additional recordings were obtained. Figure 7*B* shows a recording made a week after the hemorrhage and while the patient was receiving antispasmodics. This shows a D. C. level of —7 mv., and 3 cpm activity of greater than normal amplitude. Medicines were then discontinued, and three days later the recording shown in Figure 7*C* was obtained. That morning the patient had a return of symptoms similar to those he had experienced a week before his hemorrhage; he felt nauseated and had discomfort in the epigastrium but did not vomit. This record illustrates several of the fundamental variations seen in electrogastrographic recordings: The average D. C. level is now + 18 mv., large slow waves of period 3 to 5 minutes are visible, and 3 cpm waves of 1 mv. amplitude are superimposed along with higher frequency spike components.

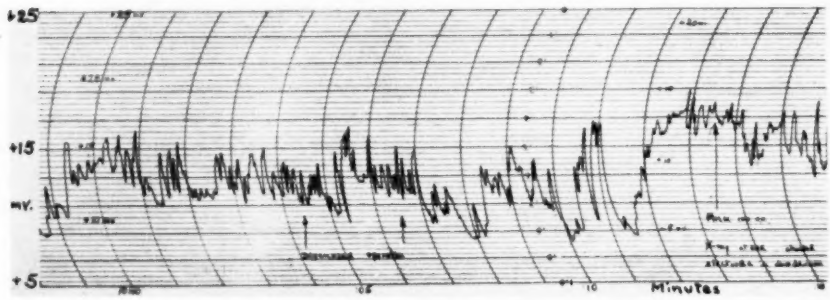


Fig. 8.—Preoperative recording of acute gastric erosions.

The question now arises, can gastritis and ulcer be differentiated? Not enough recordings are available at present to answer this question, but Figure 8 illustrates the possible aid the electrogastrogram may be in differential diagnosis. This patient was admitted because of hematemesis. There was a previous history of duodenal ulcer, but roentgenological investigation showed no definite lesion. A subtotal gastric resection was performed, and the patient was found to have acute gastric erosions. The mean D. C. level was + 10 mv., and the amplitude and frequency of the waves were increased.

D. Response to Drugs.—The response to certain drugs has been investigated, and illustrations will be presented in a future publication. When histamine was administered, the electrogastrographic recording became more positive, as would be expected with increased HCl secretion and consequent increase of the diffusion potential. This agrees with the findings of Rehm,[10a] Rice,[11] and Sawyer.[12d] Whether or not the gastric electromotive force itself changes is not known. An increased motility has also been noted in some cases.

Recordings have also been taken during the administration of insulin, and the usual response has been a change in the positive direction followed by increasing

neuromuscular activity. An example of this is shown in Figure 9, which is a portion of a recording indicating the activity 20 minutes after 5 units of insulin was administered intravenously; it also shows the response to drinking orange juice.

When 5 mg. of bethanechol chloride is given orally to patients with peptic ulcer, the response has been found to depend on the stage of therapy. If there has been no treatment, or if therapy has been discontinued for several days, the A. C. components show a marked increase. On the other hand, if the patient has been receiving antispasmodics for some time, the stimulation is negligible.

E. Carcinoma.—The patterns obtained from cases of gastric carcinoma have not been so distinctive as those from peptic ulcer. There appears to be some inhibition of the electrical activity, and some records have shown occasional random spiked potentials suggestive of the recordings obtained by Fukushima and coworkers.[9] This is being studied further.

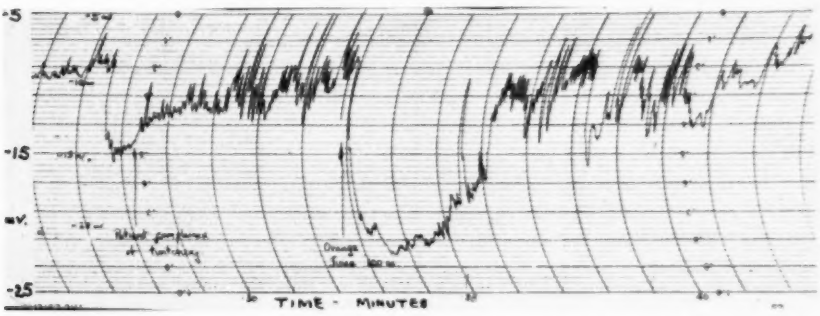


Fig. 9.—Recording of response to intravenous injection of 5 units of insulin.

F. Psychic.—No attempt has been made to evaluate the response to psychic stimulation, but on three occasions distinct changes were observed in the recordings when patients discussed subjects of considerable emotional significance to them.

COMMENT

From these records it is seen that clinical electrogastrographic recordings showing significant changes in certain diseases and in response to drugs are obtainable. Because of the complex nature of this record, compared with that of the electrocardiogram, for example, it will undoubtedly be some time before reliable interpretations will be made. That it is important to show both the wave form and the D. C. component of the electrogastrogram has been shown by the recordings of patients with peptic ulcer. Figure 10 shows the mean value of the D. C. level for normal patients and for those with peptic ulcer. Though the number of patients is insufficient for statistical analysis, these observations are suggestive.

The average value of D. C. level found in these observations is about half that found by other investigators [29] and none of these observers has reported positive

29. Footnotes 11 and 12*b-d*.

values such as we have found in certain cases of gastric pathology. Some of these differences may be accounted for by the variation in the time at which the recordings were made; certainly differences in electrodes and liquid junction potentials will cause our results to be more positive than those obtained using N/10 HCl electrodes. The fact that zero appears to be the dividing line between normal and abnormal in these recordings is of no particular significance because the zero is arbitrary; the important factor is the difference in level. The D. C. range observed has been from — 90 to + 50 mv. All the positive recordings (i. e., above + 5 mv.) have been from patients with gastric pathology, but in many pathological cases there were negative levels down to — 70 mv.

Whether or not it would be advantageous to eliminate the liquid junction potential between the electrode and gastric secretion is debatable. It would eliminate one variable, but it provides a good indication of the change of rate of HCl secretion. Rehm and others [30] have shown quantitatively that the change of potential difference

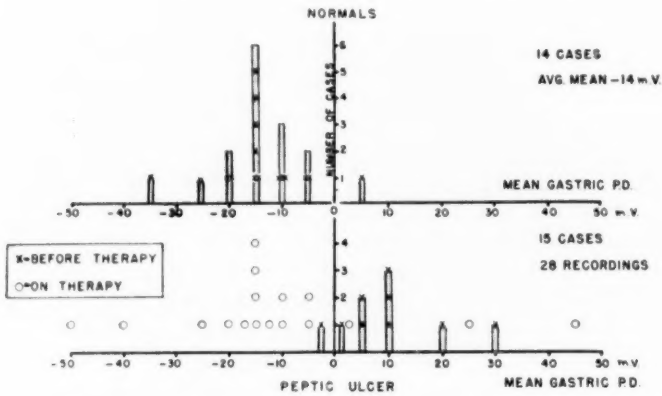


Fig. 10.—Distribution of average D. C. levels in normal cases and in cases of peptic ulcers before therapy.

associated with increased secretion is not due to a change of gastric electromotive force but may be accounted for by diffusion potentials. Also, it would be a difficult problem, for although saturated KCl would reduce the diffusion potential to a minimum, it would make the swallowing of the electrode most distressing.

The possibility that the gastric electromotive force is the mechanism by which HCl is secreted has been considered by several authors.[31] Rehm [32] concludes that it is not capable of supplying the amount of energy required but suggests that it may control the reaction. Keller and Pisha,[31a] influenced by the work of Crane and his co-workers,[1] believe it causes secretion of HCl by electrolysis. With the use of vital dyes they have observed sodium passing from the gastric contents into

30. Rehm.[10j] Hokin.[22]

31. (*a*) Keller, R., and Pisha, B. V.: Rev. Gastroenterol. **14**:495, 1947. (*b*) Crane.[1] (*c*) Rehm.[10a] (*d*) Rehm.[10j] (*e*) Coy.[23]

32. Rehm.[10g, 1]

the parietal cells and chlorine passing from the cells into the secretions. It is unlikely that electrolysis is the explanation; if it were, H^+ should also be attracted to the negative mucosa and should be involved in any reaction taking place, rather than Na^+ which has a higher discharge potential—witness the electrolysis of NaOH with the liberation of H_2 at the cathode. Admittedly, they assert that the sodium moves as an atom and not as an ion, but this does not fit in with the presently accepted theory of ionization.

Davenport [33] describes a reverse chloride shift with Cl^- passing from the blood into the gastric mucosa, and postulates the formation of H_2CO_3 from CO_2 under the influence of carbonic anhydrase. Hollander [34] thinks that the actual mechanism of secretion must be a simple inorganic reaction, and he has observed that HCl secretion is practically isotonic with tissue fluids. Gray and others [35] have shown that the rate of HCl secretion is roughly proportional to the consumption of glucose by the mucosa.

Possibly the reaction is a combination of all these factors—the utilization of glucose for energy, the absorption of some Na^+ in exchange for H^+ coupled with the secretion of Cl^-, in such proportion that isotonicity and electrical neutrality of the secretion are maintained.

Whatever the mechanism, the metabolic activity of the mucosal cells is the source of energy for HCl secretion, and the mucosal electromotive force is undoubtedly a reflection of the sum total of this vital activity. It is not logical to assume that this electromotive force is the result of a single phenomenon; there are probably at least three factors involved. Some of the electromotive force may be the result of an oxidation-reduction reaction carried on by the cells and may vary with secretory activity; a portion must be due to diffusion potential caused by different ionic concentrations between the mucosa and secretion, and some is probably due to a phase-boundary potential [36] between the cell membrane and the secretion. The role of the mucous cells must also be considered, for potential differences are found across other secreting mucosae in the body. It is hardly worth while calculating the magnitude of the diffusion potential between the cells and the gastric secretion on the basis of physical chemistry, for Osterhout [37] has shown that apparent ionic mobilities are not constant but vary with the species of cell with which the solution is in contact. Whether this resultant electromotive force is a contributing factor to HCl secretion (or gastric secretion in general) by such a mechanism as electroosmosis is speculation, but a transport mechanism based on electrolytic theory is most unlikely, considering that both positive and negative ions have to be moved in the same direction.

Concerning the wave forms, it is again noted that our recordings are remarkably similar to those obtained years ago by Alvarez,[3] and recently by Fukushima and co-workers,[9] and by Schuster and Schmucker [8] in spite of the different electrodes used. This suggests that any artefact caused by the electrodes cannot be great.

33. Davenport, H. W.: Gastroenterology **1**:383, 1943.

34. Hollander, F.: Gastroenterology **1**:401, 1943.

35. Gray, M.; Hokin, L. E., and Rehm, W. S.: Abstracted in Am. J. Physiol. **151**:380, 1947.

36. Abramson, H. A.: Electrokinetic Phenomena and Their Application to Biology and Medicine, American Chemical Society Monograph Series, No. 66, New York, The Chemical Catalog Company, Inc., 1934. Beutner.[18]

37. Osterhout, W. J. V.: Physiol. Rev. **16**:216, 1936.

The slow waves of period 3 to 5 minutes have not been described previously and may be associated with nausea. Goodman's [13] recently modified equipment appears to produce recordings similar in all respects to ours, and comparison is eagerly awaited.

CONCLUSIONS

The electrogastrogram will be useful in clinical diagnosis because it provides a continuous record of stomach motility and changes of secretory rate; this method should further our knowledge of gastric physiology and pathology. Although the interpretation is still in its infancy, reproducible recordings have been obtained and several fundamental phenomena have been recognized.

The characteristic changes seen in cases of peptic ulcer are striking, and the electrogastrogram could probably be used as a screening test before roentgenography. The progress of therapy in these cases may also be followed. Standard tests for secretion and motility may be developed on the basis of the response to such drugs as histamine and bethanechol chloride. The effect of new drugs may also be evaluated.

It is hoped to be able to show characteristic changes in cases of gastric carcinoma; this is still being investigated. Study of the "no-man's-land" between normal and organic disease should be most profitable, and it may be possible to detect electrical changes before symptoms of disease appear. Since the stomach often reflects the presence of disease elsewhere in the body, this phase of investigation may prove illuminating.

The cause of the gastric electromotive force has been discussed, and it is suggested that the D. C. component must be the combination of at least three factors: (1) metabolic activity of mucosal cells, which is probably an oxidation-reduction reaction; (2) diffusion potentials; (3) a phase-boundary phenomenon.

SUMMARY

A review of the measurement of the gastric electromotive force, the factors influencing it, and the technical difficulties in recording it has been presented.

The normal electrogastrogram has been shown to consist of a D. C. component with superimposed A. C. components, chiefly 3 cycles per minute (cpm) but with higher frequencies up to 12 cpm.

Large slow waves of period 3 to 5 minutes have been observed under abnormal conditions which may be associated with nausea.

An increased negativity has been observed in response to drinking, and an explanation for this has been given.

Characteristic recordings of peptic ulcer and the response to therapy have been shown.

The response to certain drugs has been recorded.

The nature of gastric electromotive force and the mechanism of HCl secretion have been discussed.

The patients at Queen Mary Veterans Hospital cooperated in this study.

Dr. J. F. Davis, of the Allan Memorial Institute, and Dr. H. H. Jasper, and Mr. L. H. Geddes, of the Montreal Neurological Institute, contributed valuable suggestions and criticisms.

DISCUSSION

DR. J. W. SCOTT, Toronto, Canada: The work that Drs. Morton and Martin have described shows very interesting physiological problems which as yet have not been solved. However, they have taken the empirical findings and, without a complete explanation, have shown that those findings will change with the presence of pathological lesions.

They have introduced, and emphasized, a possible clinical tool that will ultimately have a much more complete explanation. Meanwhile, if their work is confirmed and expanded, it may prove of considerable value in the diagnosis and mangaement of peptic ulcers and, perhaps, even cancer.

EXPERIMENTAL METHOD FOR AN EVERTING END-TO-END ANASTOMOSIS IN THE GASTROINTESTINAL TRACT

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AND
WILLIAM M. TUTTLE, M.D.
DETROIT

IN WORK done previously in our laboratory on the blood supply of the esophagus, approximately 25% of the dogs on which resections were made were seen to retch on solid food two weeks after conventional esophageal anastomoses.[1] In later attempts to transplant sections of viable intestine and other tissues into surgical defects in the esophagus the regurgitation of food and fluid was such a prominent feature of the postoperative course that resultant malnutrition usually jeopardized the end-result. The edema and induration often persisted with a high degree of temporary stenosis for 10 to 14 days. It is largely for this reason that many surgeons rely on the weaker, more time-consuming side-to-side anastomosis in small-bowel resections. The conventional anastomosis occasionally presents the surgeon with a serious complication elsewhere, in the form of the nonfunctioning anastomosis.

These problems prompted a review of suture and anastomotic techniques in the hope that a satisfactory method could be found which would insure a patent lumen during the early phases of the healing process. Such an anastomosis would have to fulfill the prerequisites and conform to the high standard of functional results of those methods accepted and generally employed today, but in addition should afford a high degree of patency throughout the healing phases. The inverted tissue flange in the conventional suture methods represents the organic structure which, with edema and induration, gives sufficient bulk to form a stenosing and often obstructing diaphragm at the site of anastomosis. It is therefore apparent that a suture method resulting in the smallest intraluminal diaphragm is the most likely to present a patent end-result. Spivack states:[2]

> The real knowledge of intestinal suture came with the contributions of Jobert and Lembert. The latter showed that good union will follow if the serous surfaces of the loops are brought

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

From the Department of Research Surgery, Harper Hospital and Herman Kiefer Hospital, and Department of Surgery, Wayne University College of Medicine.

1. Shek, J. L.; Prietto, C. A.; Tuttle, W. M., and O'Brien, E. J.: Experimental Study of Blood Supply of Esophagus and Its Relation to Esophageal Resection and Anastomosis, J. Thoracic Surg. **19**:523-533, 1950.

2. Spivack, J. L.: The Surgical Technic of Abdominal Operations, Chicago, S. B. Debour, 1936, p. 127.

together. Later experiments, however, have shown that good union of the parts of a stomach or bowel wall will follow also when the corresponding layers are sutured together, namely serosa to serosa, muscularis to muscularis and mucosa to mucosa, and that the real holding layer, which gives firm strength to the suture line is the muscularis-mucosa of each side. This must be included in the suture line. The serosa in fact serves only as the plastic material which prevents leakage of the gastro-intestinal contents into the free peritoneal cavity.

Therefore it was felt that an everting method which would permit an anatomical end-to-end approximation of the individual layers of the structure to be anastomosed, leaving no intraluminal diaphragm, would best fulfill the requirements. An end-to-end approximation has been employed experimentally by Butsch [3] and others [4] with apparent success.

An everting suture method was first described by Heister [5] in 1763. This method was described as an "everting thru and thru suture designed to approximate wounds where the tissues can not relax." This early quotation indicates one advantage of the suture method—additional strength. Recently similar suture techniques have been used in vascular surgery where the necessity of a patent lumen and a non-leaking strong anastomosis has proved them most effective. Several methods and types of anastomosis were attempted. The technique settled upon for use in the esophagus and employed thus far with only a few modifications consists of the following simple method.

TECHNIQUE

The technique consists of interrupted full-thickness, through-and-through, everting mattress sutures of No. 000 or 0000 silk. A second layer of running No. 0000 silk suture is used to approximate muscularis and serosal layers (Fig. 1).

The first layer of mattress sutures everts a full thickness rim or flange of tissue approximating mucosa to mucosa for the circumference of the anastomosis (Figs. 2 and 3). The sutures are placed approximately 4 mm. from the cut ends of the wall, causing the everted flange to be 2 to 3 mm. in depth. A smaller bite is taken of the mucosa than of muscularis and serosa. This enables a better approximation of the tissues by the second layer of running sutures which incorporate only the muscularis and/or serosa (Fig. 4).

The layer of mattress sutures has been sufficient for satisfactory healing, as in most of the anastomoses carried out thus far we have used the second layer on only one-half the circumference to permit comparison. However, the second layer adds an additional safety factor, decreases the number of adhesions at the site of anas-

3. Butsch, W. L.: Personal communications to the author.

4. (*a*) Trueblood, D. V.: Intestinal Anastomosis Using Simplified Basting Stitch Method, West. J. Surg. **40**:654-658, 1932. (*b*) Price, P. B.: Stress, Strain and Sutures, Ann. Surg. **128**:408-421, 1948. (*c*) Connell, F. G.: Intestinal Sutures: Some Old, Some Not So Old, and a New One, Philadelphia Month. M. J. **1**:37-56, 1899. (*d*) Connell, F. G.: Intestinal Suture, Perforating and Knotted in the Lumen, Surg., Gynec. & Obst. **4**:534-536, 1907. (*e*) Connell, M. E.: An Experimental Contribution Looking to an Improved Technique in Enterorrhaphy, Whereby the Number of Knots is Reduced to Two or Even One: This Suture is Applicable in Pylorectomy, Gastroenterostomy, Anastomosis, Circular and Oblique Enterorrhaphy, Med. Rec. **42**:335-337, 1892. (*f*) Maunsell, H. W.: A New Method of Intestinal Surgery, Am. J. M. Sc. **103**:245-257, 1892. (*g*) Senn, N.: Enterorrhaphy: Its History, Technique and Present Status, J. A. M. A. **21**:215-235, 1893. (*h*) Halsted, W. S.: Circular Suture of the Intestine: An Experimental Study, Am. J. M. Sc. **94**:436-461, 1887. (*i*) Halsted, W. S.: Intestinal Anastomosis, Bull. Johns Hopkins Hosp. **2**:1-4, 1891.

5. Heister, cited by Gould, A. H.: The Technic of Operations upon the Intestines and Stomach, Philadelphia, W. B. Saunders Company, 1906.

tomosis, and generally improves the final appearance of the anastomosis. It also tends to ease the esthetic sense of the surgeon who is disturbed by protruding bits of mucosa. It is not necessary to cover all the mucosa, however, to have a satisfactory anastomosis.

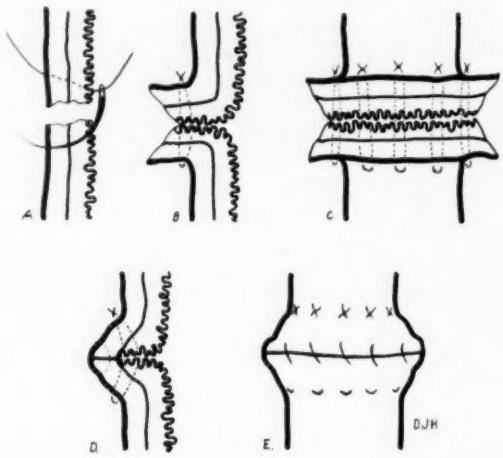

Fig. 1.—Diagram shows mechanical steps in anastomosis. *A* demonstrates needle passing in oblique line through wall of sutured structure, taking longer bite of serosal surface and shorter segment of mucosal surface. *B* and *C* represent appearance of approximated segments. Former is wall of structure in cross section; latter shows both segments brought together by everting mattress sutures. *D* and *E* represent finished anastomosis after second layer has been placed.

Fig. 2.—This demonstrates first and everting layer in posterior wall of dog's esophagus. Mucosal layer is approximated without intraluminal diaphragm.

DISCUSSION OF TECHNIQUE AND RESULTS

This method of anastomosis was first applied to the esophagus, and the initial eight anastomoses were carried out without complication. In each case the animal was fed its regular diet consisting of prepared dog food within the first 12 hours after operation. As the dogs tolerated this early feeding well and in only one instance were seen to regurgitate, we placed food in the pen, and some dogs ate heartily

within a few hours. The animals were carefully observed at feeding time, and only one dog was seen to regurgitate a portion of the feeding on two occasions. This was in marked contrast to the experiences with many esophageal anastomoses of the

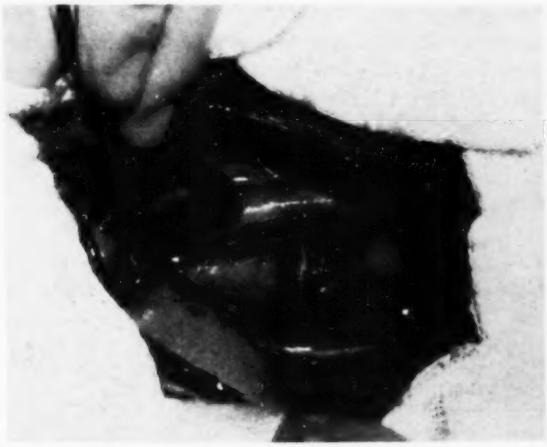

Fig. 3.—Everting layer is complete. Note everted flange of tissue and wider than normal lumen at site of anastomosis.

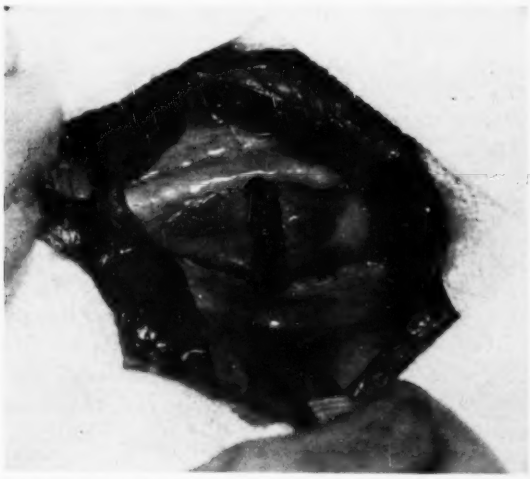

Fig. 4.—Second layer is complete and anastomosis is finished. Sutures actually sustain anastomosis in position of patency.

conventional type, in which regurgitation of a varying degree was a usual sequela lasting from 7 to 10 days in all the dogs.

Our attention was then changed to the intestinal tract, as here we had the problem of intraluminal pressure, leakage, and contamination, which would be a more discerning test of the integrity of the method of anastomosis.

There have been 18 anastomoses in all, performed high in the small intestine by this technique, with two deaths due to leakage and peritonitis. These occurred, however, in the first and third dogs in which the intestinal anastomosis was performed. Both deaths were attributed to technical errors which occurred in the process of improving the method. In the first fatality, the anastomosis was started at the antimesenteric border of the intestine, so that the final mattress sutures were placed in the mesenteric portion of the intestinal wall, where poor visibility and the mechanical difficulty of placing sutures contributed to a leak at that point. We now place the first sutures at the mesenteric portion of the bowel wall, and have had no further difficulty with this problem. The second complication was a leak which occurred as the result of tying two adjacent sutures sufficiently tight to cut through the bowel wall. This is a technical error which may occur in any method if sutures are improperly tied.

To date, three gastroesophageal anastomoses have been carried out in which the lower half of the esophagus was removed, the stomach brought up through the diaphragm, and an anastomosis made at a different site on the fundus of the stomach. The first of the dogs thus treated died 18 hours after operation because of unrecognized excessive blood loss from the diaphragm. The second dog died five days after operation with a chylothorax. The last dog died of pneumonia six weeks after operation after a change in habitat to less favorable quarters. In all dogs, however, the anastomosis was intact, and there was no evidence of leak.

We believe the results thus far to be good, considering that in only the past 6 to 10 anastomoses has the technique become sufficiently standardized to be removed from the category of modifications of the original technique.

PATHOLOGY OF HEALING

In order to determine whether healing would occur in an acceptable manner, anastomoses were removed from the animals at four-day intervals until the 16th day and at intervals of several weeks thereafter. Several dogs had two or three anastomoses performed on the small intestine. The one removed at the desired interval was replaced by a similar end-to-end anastomosis. It was noted that with each subsequent anastomosis there seemed to be a greater tendency for the formation of adhesions around the suture line. On the whole, however, there was no greater number or density of adhesions around a given anastomosis carried out by the everting technique than was found on a small group of Mann-Williamson dogs on which anastomoses were performed by the conventional methods during the same interval. There was no evidence of obstruction or distention proximal to the suture line as indicated by increased diameter of the bowel or of its lumen. Intraluminal sloughs and granulations did not occur in the everted anastomoses (Figs. 5 and 6). Pinhead-sized granulations were found in two unsupported esophageal anastomoses where a suture had been pulled out and left dangling in the lumen.

Microscopically, healing seems to occur quite similarly to that noted in the conventional anastomosis, except that the mucosal layer at no time is in jeopardy. We believe that the integrity of this layer may actually decrease the amount of local induration by presenting a healthy barrier to the intestinal contents. It was unusual to find intestinal parasites fastened to the suture line, as often seen in the conventional anastomosis. The intestinal wall distal to the suture line appeared to be sealed promptly by a layer of exudate, which soon supports fibroblastic activity

with resultant firm healing. No abscess of any size was demonstrated in the intestinal wall or the peritoneal cavity, except in those animals that died early in the experiments. The anastomoses examined after several months had flattened in the reverse of the process in conventional anastomoses.

The conclusion gained from examination of the microscopic sections of the healing anastomosis was that the everted flange of tissue supplies an adequate valve

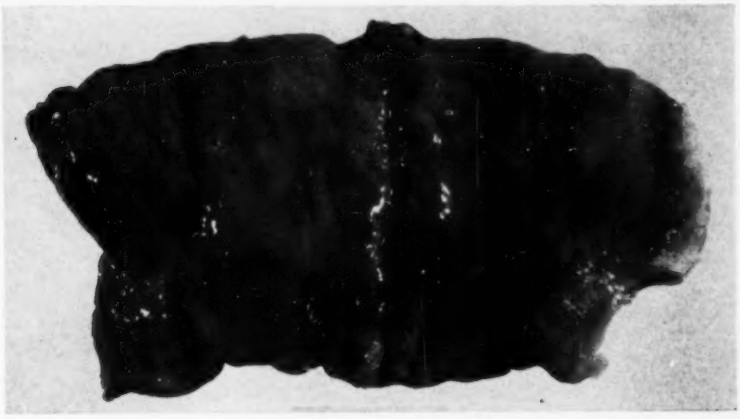

Fig. 5.—Absence of granulations and grossly satisfactory healing are demonstrated in opened fresh anastomosis removed after eight days.

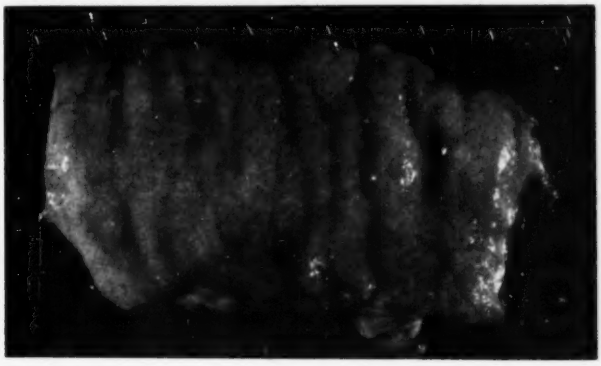

Fig. 6.—Anastomosis is shown at 20 days.

to prevent leakage, and the stronger suture method assures adequate approximation while end-to-end healing takes place (Figs. 7 and 8).

ADVANTAGES OF EVERSION TECHNIQUE

The advantages seen in this method of anastomosis are:

1. The unquestionable patency of the lumen despite the inevitable edema and induration. This has been demonstrated by complete absence of obstructive signs

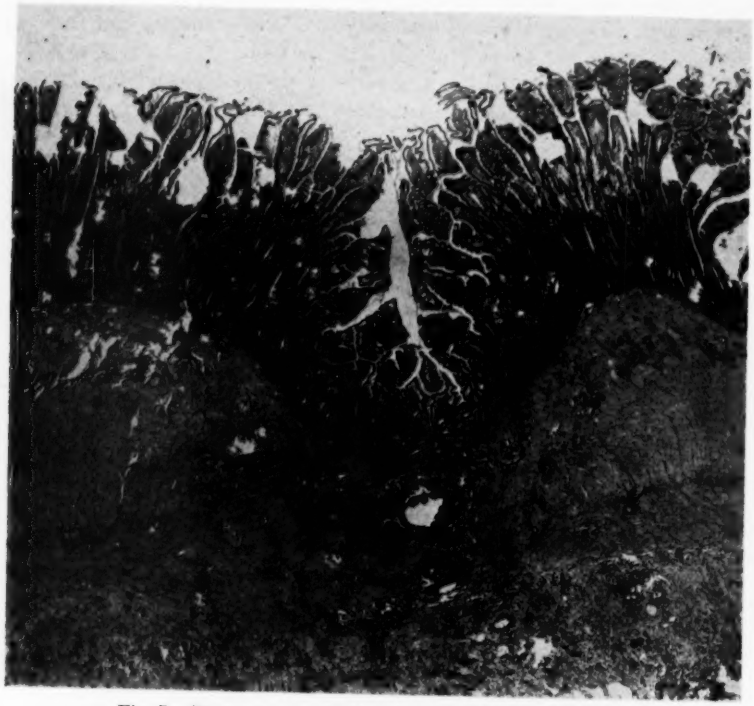

Fig. 7.—Photomicrograph shows healing process at 16 days.

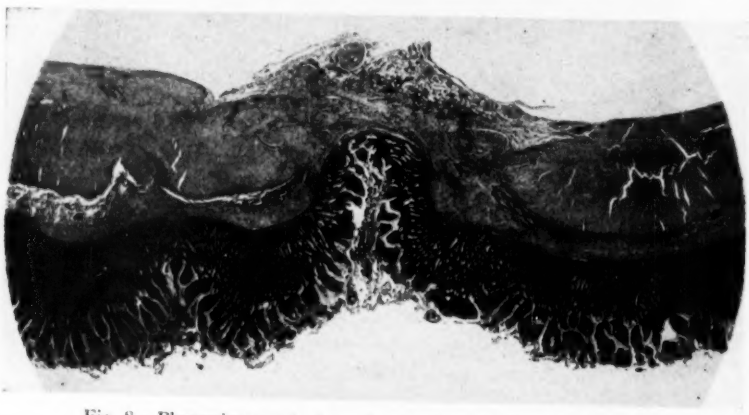

Fig. 8.—Photomicrograph shows intestinal anastomosis at 12 weeks.

in any of the animals thus far. It is most dramatically demonstrated in the esophagus which normally is collapsed, for here the lumen at the anastomosis is patent even though the walls of the remaining structure are collapsed.

2. This larger lumen results in a lower hydrostatic pressure at the site of the anastomosis than elsewhere in the lumen and therefore has less tendency to leak.

3. It is a strong anastomosis, as all the layers are incorporated by a single suture. Therefore greater longitudinal and intraluminal pressures can conceivably be withstood.

4. There is no greater chance of leak at the line of anastomosis, since there is an everted flange of tissue which is approximated for 3 to 5 mm. to act as a seal against leakage.

5. There are no sutures compromising the blood supply to any portion of the intraluminal mucosa. No intraluminal slough has been demonstrated, and intraluminal granulation and ultimate cicatrix formation with stenosis is therefore unlikely to occur.

6. Hemostasis in the line of anastomosis is easily obtained and need be of little concern by virtue of the fact that the cut edges are at all times everted for inspection.

7. It is a relatively simple technique and easily performed. The problems of corners, angles, and desirable locations for beginning and ending continuous sutures are largely eliminated.

8. Any type of end-to-end anastomosis has proved stronger than lateral anastomoses.[4b]

INDICATIONS FOR USE

The most appropriate location for the application of this suture technique appears to be in esophageal anastomoses, for the esophagus has no serosa, requires additional strength because of the suspended weight it may be called upon to support, and is especially prone to cicatricial stenosis following conventional anastomoses. The method may be specifically indicated in esophageal atresias of congenital origin and for replacement of localized acquired cicatricial lesions. In the intestinal tract, a congenital atresia again seems to be a prime indication because of the large proximal segment which must be anastomosed to a disproportionately small distal segment, with a functioning anastomosis meaning the difference between success and failure. Elsewhere in the gastrointestinal tract this method of anastomosis would afford the surgeon doing only an occasional resection a safe, easy, and quick method, and the assurance that he will have a strong, functioning anastomosis throughout the entire healing phase.

COMBINED ABDOMINOENDORECTAL RESECTION

Technical Aspects and Indications

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ROCHESTER, MINN.

THIS METHOD of resecting the upper part of the rectum and rectosigmoid was reported in 1948.[1] The anastomosis is achieved not by suturing, but by invaginating the sigmoid through the intact anal canal. The particular advantage of the procedure is that fecal continence, rather than only a sphincter capable of contracting, is preserved. At the time of the previous report seven patients had been treated. The increased experience with 41 patients serves as the basis for the present report.

Resections of the rectum with restoration of continuity of the bowel antedated radical amputations of the lower reaches of the bowel by almost a quarter of a century. Procedures of the Kraske[2] type, in which continuity is restored by circular suture, proved quite unsatisfactory because of leakage of the anastomosis. Hochenegg,[3] three years after the introduction of the Kraske method, attempted to avoid anastomosis by suturing, by use of the pull-through method which has since been associated with his name. Within the next 10 years, virtually all possible methods of restoring continuity of the bowel at this level were attempted or described. Suturing from within the lumen of the bowel, the method revived by Swenson in his procedure for megacolon, was first described by Maunsell[4] in 1892, and later by Weir,[5] in 1901. The Murphy button was employed for the anastomosis at approximately the same time. The use of a tube to intussuscept the sigmoid into the rectum was not described until 1910. However, as Balfour[6] pointed out in his description of the technique, the method had been used for some time.

From the Division of Surgery, Mayo Clinic.

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March, 1952.

1. Black, B. M.: Combined Abdominal Endorectal Resection: A Surgical Procedure Preserving Continuity of the Bowel, for the Management of Certain Types of Carcinoma of the Midrectum and Upper Part of the Rectum, Proc. Staff Meet., Mayo Clin. **23**:545-554 (Nov. 24) 1948.

2. Kraske, P.: Zur Exstirpation hochsitzender Mastdarmkrebse, Verhandl. deutsch. Gesellsch. Chir. **14**:464-474 (April) 1885.

3. Hochenegg, J.: Die sacrale Methode der Exstirpation von Mastdarmkrebsen nach Prof. Kraske, Wien. klin. Wchnschr. **1**:254-257 (June 14) 1888.

4. Maunsell, H. W.: A New Method of Excising the Two Upper Portions of the Rectum and the Lower Segment of the Sigmoid Flexure of the Colon, Lancet **2**:473-476 (Aug. 27) 1892.

5. Weir, R. F.: An Improved Method of Treating High-Seated Cancers of the Rectum, J. A. M. A. **37**:801-803 (Sept. 28) 1901.

6. Balfour, D. C.: A Method of Anastomosis Between Sigmoid and Rectum, Ann. Surg. **51**:239-241 (Feb.) 1910.

All such methods were gradually abandoned in English-speaking countries after the introduction of combined abdominoperineal resection by Miles [7] in 1908. This was not so in Central Europe and the Scandinavian countries, where the Hochenegg pull-through procedure has been employed continuously since its introduction in 1888. Babcock [8] is clearly responsible for the renewal of interest in such procedures in English-speaking countries. Although his surgical procedure was described in 1932, it was not until some ten years later, after surgery of the colon had become somewhat safer, that such operations were more widely attempted.

The present procedure is the result of efforts to achieve better fecal control than was possible after operations in which only the sphincters were saved. In earlier operations in which I attempted to preserve continence, the sphincters which were preserved were sectioned in the midline posteriorly, the perianal skin was sacrificed, and the end of the sigmoid was left protruding well beyond the sphincters. Fecal control proved so poor that the procedure was progressively modified from case to case until ultimately all of the anal skin was retained, the sphincters were preserved intact along with the levators, and the mucosa of the sigmoid was carefully sutured to the skin of the anus at the level of the sphincters. The operation, as it had evolved to this stage, was similar in all respects to the procedure described by Roux [9] in 1927. After the Roux procedure, the anus appears normal and there is little tendency for stenosis if all of the anal skin has been saved. Such tendency to stricturing as there is can be easily overcome by having the patient insert a finger through the sphincter from time to time. The sphincter has definite tone and can be promptly and strongly contracted at will. Unfortunately, even in cases in which the anatomic result was excellent, the patients had no vestige of normal control.

It was reasoned that failure to achieve control could have been due to any or all of the following factors: lack of an internal sphincter, trauma to the external sphincter, denuding of the external sphincter, or sacrifice of the mucous membrane or muscular layers, or both, of the distal part of the rectum. By division of the bowel a short distance above the dentate margin, all structures which might contribute to control would be preserved intact. Somewhat arbitrarily, the decision was made to divide the rectum approximately 3 cm. above the dentate margin. Cunéo, Sénèque, and Zagdoun [10] included all the essential features of abdominoendorectral resection in an operation reported in 1934. The bowel was sectioned through a dorsal approach, however, and not endorectally. In some cases the mucous membrane was stripped from the rectal stump, while in others it was retained. Bergeret and Livory [11] described a similar technique in 1940. The bowel was divided while the abdominal and peritoneal cavities were still open. The method of intussuscepting the sigmoid through the anal canal was somewhat awk-

7. Miles, W. E.: A Method of Performing Abdomino-Perineal Excision for Carcinoma of the Rectum and of the Terminal Portion of the Pelvic Colon, Lancet **2**:1812-1813 (Dec. 19) 1908.

8. Babcock, W. W.: Carcinoma of the Rectum; One-Stage Simplified Proctosigmoidectomy with the Formation of a Perineal Anus, S. Clin. North America **12**:1397-1400 (Dec.) 1932.

9. Roux, C.: Resection of the Rectum and Colon Through the Anal Sphincter: Post-operative Results, Practitioner **118**:284-293 (May) 1927.

10. Cunéo, Sénèque, and Zagdoun: Sur un nouveau procédé d'extirpation du rectum par voie périnéale avec conservation du sphincter, J. chir. **44**:1-11 (July) 1934.

11. Bergeret and Livory: La résection recto-sigmoïdienne avec intubation colo-rectale, J. chir. **56**:209-221 (Oct.) 1940.

ward. In both procedures, sutures were used between the rectal stump and the sigmoid to insure holding of the invaginated bowel in place. It is quite evident that abdominoendorectal resection involves no new conceptions.

TECHNIQUE OF OPERATION

Abdominal Phase.—The abdominal incision, the mobilization of the rectum and rectosigmoid from the hollow of the sacrum, and the division of the superior hemorrhoidal or inferior mesenteric vessels are carried out exactly as in combined abdominoperineal resection. As with the usual sphincter-saving procedure, there must be enough viable bowel to reach beyond the sphincter. In a limited number of cadavers, the distance from the promontory of the sacrum to the perineum was found to be approximately one-third to one-half greater than the distance from the promontory to the pubis. By use of the latter measurement, the length of the segment of bowel necessary to reach the perineum may be estimated with some accuracy. Attention must be paid to the preservation of the arterial supply of the preserved sigmoid and descending colon. In my experience the securing of sufficient length of adequately vascularized bowel is the most difficult part of the procedure. As judged from the emphasis given the difficulty by Mandl [12] and by Bacon,[13] the problem is probably inherent in pull-through procedures. The adequacy of the circulation should always be verified by demonstrating active arterial bleeding when a small vessel is opened at the level at which the sigmoid is to be subsequently divided. This level is marked by tying a stout ligature around the bowel.

Distally, the dissection is carried to the levators behind and to the tip of the prostate or perineal body anteriorly. After complete mobilization of the rectum and rectosigmoid, a second heavy ligature is tied around the bowel somewhat above the level at which the rectum subsequently is to be divided. If the lesion is bulky or if peritonealization cannot be easily achieved, the bowel may be resected at this stage. The cut end of the sigmoid and that of the rectum are inverted and the two ends are tied together. The endorectal phase, in this case, is identical with that in which the bowel has not been resected. Hemostasis must be meticulous since the perirectal region is not again exposed. A new peritoneal floor is constructed in the usual fashion after the mobilized bowel has been packed into the pelvis (Fig. 1). The abdomen is closed without drainage.

Except for the problem of an adequate length of vascularized sigmoid, the procedure to this point is easier and far more quickly accomplished than the abdominal phase of combined abdominoperineal resection, since no colonic stoma is necessary. It is usually possible to reperitonealize without resecting bowel. In these cases, there is no possibility of contamination unless the lesion has perforated. This is perhaps less of an advantage at present with current methods of preparing the bowel than it was in the past.

Endorectal Phase.—This part of the procedure is simple and quickly finished. With the patient in the lithotomy position, the sphincter is dilated and the short rectal pouch, limited above by the distal ligature around the bowel, is cleansed.

12. Mandl, F.: Technique and Results of Primary and Secondary Pull-Through Operation After Removal of Tumors of the Rectum and Rectosigmoid, Surgery **18**:318-335 (Sept.) 1945.

13. Bacon, H. E.: Evaluation of the Babcock Technic of Proctosigmoidectomy for Cancer of the Rectum, Tr. Am. Therap. Soc. **45**:64-66, 1945-1946.

The constricted part of the bowel is grasped with a hemostat, and the rectal stump is everted. By use of diathermy, a circular incision is made through the entire thickness of the wall of the bowel at the selected level, usually 3 to 4 cm. above the dentate margin (Fig. 2). After complete transection of the rectum the

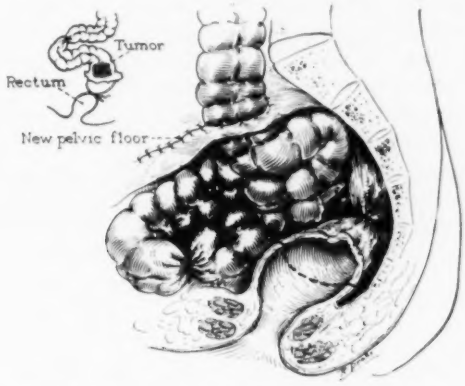

Fig. 1.—The lower part of the sigmoid and the rectosigmoid and rectum have been mobilized from the hollow of the sacrum, and ligatures have been applied, as shown in the inset, roughly isolating the segment of bowel to be resected. A new peritoneal floor has been constructed, after the mobilized bowel has been packed into the pelvis.

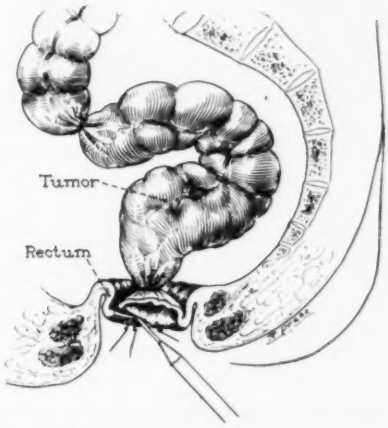

Fig. 2.—Endorectal phase of the operation. The bowel is grasped with a hemostat (not shown) at the point at which the ligature was applied. The rectum is sectioned, by means of diathermy, 3 to 4 cm. above the dentate margin.

previously mobilized bowel is carefully drawn down through the intact anal canal until the higher ligature around the bowel, marking the site of viable vascularized sigmoid, is well beyond the sphincter (Fig. 3). Considerable care should be exercised not to avulse the blood supply from the lower part of the sigmoid. The preserved marginal artery or other vascular arcade forms a cord to the arc of

the bowel. Rough handling or tension can easily tear the vessel from the bowel or break the vascular arcade. If arterial bleeding high in the pelvis should suddenly develop at this stage, the abdomen and new peritoneal floor should probably be reopened to secure the bleeder and to determine the extent of devascularization. Unless a sufficient length of vascularized bowel remains, the operation may have to be completed as a Miles or Hartmann procedure.

The redundant bowel, including the segment in which the lesion is situated, is removed, leaving 4 to 5 cm. of sigmoid protruding beyond the anus (Fig. 3). The protruding bowel is held in place by suturing epiploic tags to perianal skin. No sutures are used to maintain the anastomosis, nor are any sutures placed between the rectal stump and sigmoid. The protruding sigmoid may be left occluded for a few days, or a large rectal tube may be tied into the end of the bowel. I prefer the former method, since it is less awkward, particularly after the patient is allowed up on the first or second day after operation. I usually drain the space in the

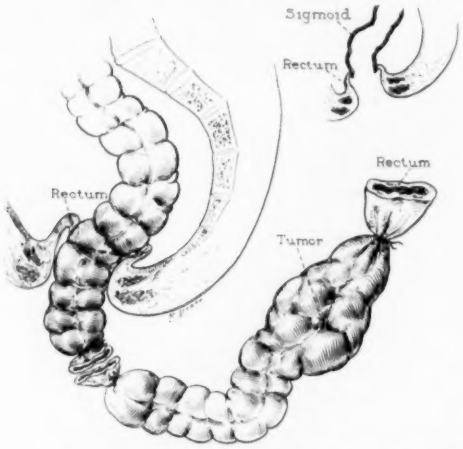

Fig. 3.—The segment of bowel to be resected is pulled through the intact anal canal until a portion of viable bowel, marked by the proximal ligature, is reached. The protruding part of the bowel is anchored in place by the suturing of epiploic tags to the perianal skin. The inset illustrates the anastomosis after amputation of the redundant section of bowel two weeks later.

hollow of the sacrum through a stab wound just anterior to the coccyx. The drains are removed as a rule 12 to 24 hours before the bowel is opened, in an effort to prevent contamination.

Removal of Redundant Bowel.—The segment of sigmoid left protruding beyond the anus is amputated in approximately two weeks. The sigmoid is divided above the level of the sphincter and well below the junction between the cut end of the rectum and the sigmoid. If union between the rectum and the sigmoid does not seem completely satisfactory, or if an opening exists between the lumen of the rectum and the perirectal space, amputation of the bowel should be postponed until healing is more advanced.

Subsequent Course.—The sphincters, which were kept dilated by the protruding sigmoid and which were redilated to permit amputation of the redundant bowel,

require from 7 to 14 days to regain their normal tone. During this interval the patient is of course incontinent. Rectal sensation, however, is present, and the return of control begins almost immediately after final amputation of the bowel. At this stage the sigmoid protrudes into the rectum much as the cervix protrudes into the vagina. There is a definite tendency for the cut end of the sigmoid to contract, and the tendency to contract continues until the epithelium of the rectum meets that of the sigmoid. Two to three months may be required for complete epithelization, during which time the cut end of the sigmoid should be dilated digitally. The dilatations may be spaced progressively further apart, depending on the tendency to stricture in the individual case. Since the strictured site is not an anastomosis, there is practically no danger of causing leakage by the dilatations.

Complete continence may be achieved within two or three weeks in some cases; in others the patient may insist on a protective pad for a month or so. How quickly continence is regained probably depends on the extent of the local inflammatory reaction and the speed with which epithelization progresses. I have attempted, at the time the bowel was amputated, to suture the two mucous membranes together. The sutures usually did not hold and probably did not speed final healing in any case. Anastomosis by inversion heals very similarly to anastomosis by suturing, and final healing probably requires about the same length of time.

COMPLICATIONS

Hospital Deaths.—There were two hospital deaths in the series. One death resulted from generalized peritonitis and the other from pseudomembranous ulcerative ileocolitis. In spite of a hospital mortality rate in this limited series comparable with that after combined abdominoperineal resection, endorectal resection seems far better tolerated by the patient. The abdominal phase is simpler and more quickly carried out, while the endorectal phase is a minor maneuver easily completed within four or five minutes. The surgical trauma is more nearly comparable to that of abdominal hysterectomy than to that of combined abdominoperineal resection.

Slough of Sigmoid.—In spite of all precautions and in spite of testing for active bleeding in all cases, some slough of the sigmoid occurred in 6 of the 41 cases. Unexpected sloughing in spite of positive evidence of adequate circulation has been alluded to previously in the literature after such procedures. I am confident that in several cases, if not in all in which slough occurred, the vascularity was adequate at the conclusion of the operation. In these cases thrombosis must have developed subsequently. In one case the bowel definitely became infarcted during a prolonged hypotensive episode due to myocardial infarction. In none of the six cases could the slough be attributed to compression by the dilated but otherwise intact sphincter.

In three of the six cases the slough did not extend proximally above the rectal stump. There was no associated infection, and the net result of the slough was merely that subsequent amputation of the bowel was not necessary. Even when gangrene develops, leakage through the wall of the bowel may be delayed several days. This interval is usually long enough to allow reasonably firm union to occur between the cut end of the rectum and the sigmoid. It is my practice to inspect the protruding sigmoid regularly. If sphacelation develops, the administration of antibiotics is continued or restarted and nothing further is done unless signs of pelvic infection develop.

The slough in three cases was associated with such infection. In each, drainage through the anus seemed quite adequate, systemic reaction was minimal, and a colonic stoma was not thought necessary. When healing occurred ultimately, the lumen proved adequate, and normal continence was regained in all three cases. It is probable that there is often sufficient reaction around the gangrenous bowel, before leakage of stool actually occurs, to wall off the region reasonably well. On the other hand, a diverting colonic stoma probably should be seriously considered in any case in which the slough extends above the level of the rectal stump, if only to speed up healing.

Pelvic Abscesses.—Pelvic abscesses or abscesses in the hollow of the sacrum develop probably less often after the endorectal operation than after combined abdominoperineal resection in which the posterior incision is closed. There is less dead space with the former procedure because much of the space between the levators and peritoneum is occupied by sigmoid. When such abscesses develop after endorectal resection, they tend to drain spontaneously through the rectal stump or the perineal stab wound. The complication developed in only two cases in the series.

Failure of Union Between Sigmoid and Rectum.—When endorectal resection was first considered, I anticipated that healing would occur only between the sectioned end of the rectal wall and the serosa of the sigmoid. It soon became evident that some inversion of the cut end of the rectum invariably occurred and that the union between the two segments of bowel was over a wider zone than had been anticipated. Unless an abscess develops, union is well established after 14 days. This was true in every case in which the operation was carried out because of carcinoma. In two cases the procedure was employed to resect the rectum and rectosigmoid because of megacolon in the adult. Parenthetically, anastomosis by suturing of the dilated and hypertrophied sigmoid to the lower part of the rectum in adults with megacolon is most unsatisfactory technically. The problem can be nicely avoided by employing a pull-through operation. In the first case of megacolon, the redundant bowel was amputated after 18 days, at a time when healing seemed well advanced. The hypertrophied bowel promptly retracted above the rectal stump and could not be brought down by traction. A diverting colonic stoma ultimately was thought necessary. Union between the sigmoid and rectum finally took place, and the stoma was subsequently closed. In the second case of megacolon in the adult the sigmoid was left protruding through the sphincter for six weeks. After this interval the union was firm and the bowel did not retract after amputation.

FECAL CONTROL

The revival of interest in preserving continence after resection of the rectum has led to reinvestigation of the mechanism of fecal control.[14] According to Gaston,[14a] stool is accommodated in the left part of the colon through a process of plastic enlargement. The only sensation associated with such colonic distention

14. (*a*) Gaston, E. A.: The Physiology of Fecal Continence, Surg., Gynec. & Obst. **87**:280-290 (Sept.) 1948; Fecal Continence Following Resections of Various Portions of the Rectum with Preservation of the Anal Sphincters, ibid. **87**:669-678 (Dec.) 1948. (*b*) Goligher, J. C., and Hughes, E. S. R.: Sensibility of the Rectum and Colon: Its Rôle in the Mechanism of Anal Continence, Lancet **1**:543-547 (March 10) 1951.

is one of gas referred rather diffusely to the lower part of the abdomen or left iliac fossa. After a certain point, a peristaltic rush carries the stool into the rectum. This immediately provokes a contraction of the external sphincter sufficient to prevent leakage and also gives rise to a desire to defecate (rectal sensation) referred to the presacral region or to the perineum. The rectal sensation lasts for a relatively short interval during which the rectum dilates to accommodate its increased contents. As the pressure falls, the necessity for the heightened tone of the sphincter disappears and the tone returns to the resting level. Since the rectal sensation reaches consciousness, and since the sensation may be disregarded or acted upon, both Gaston [14a] and Goligher [15] regard normal control as essentially a conscious process.

Goligher and Hughes [14b] have shown that rectal sensation is not abolished by sympathetic denervation of the rectum, nor by blocking the inferior hemorrhoidal nerves. They conclude, consequently, that sensation is mediated over the parasympathetic system via the pelvic and inferior mesenteric plexuses. Normal control is lost completely if all of the rectal wall is sacrificed, despite preservation of the sphincter. The sensory fibers to the lower part of the rectum are evidently preserved intact in procedures such as the present one. Whether the sensory receptors are in the mucosa or muscularis has not been established. The degree of control after the Hochenegg procedure, in which the mucous membrane is removed from the rectal stump, is apparently far less satisfactory than that after procedures in which the mucous membrane is not removed. Both Gaston and Goligher and Hughes found that control remained essentially normal in most cases in which the anastomosis was located 6 to 7 cm. above the anus, and that only colonic sensation remained in cases in which the sphincter alone was saved. In neither investigation were tests made on patients whose anastomoses were 3 to 4 cm. above the anus. Gaston made tests on one patient whose anastomosis was between 1 and 2 cm. above the anus. The findings as to rectal sensation were equivocal. However, the patient regarded himself as continent and had ample warning of imminent defecation.

Of the 39 patients who survived operation in the present series, the 2 patients with megacolon probably should be considered in a separate category, and control could not be evaluated in 1 patient whose resection was palliative and who had a previously established colonic stoma. In three other patients, the present status of control is not known. Of the 33 remaining patients, control was essentially normal in 26 and abnormal in 7. By "essentially normal" is meant that the patient regards himself as continent and that neither protective pads nor enemas are employed, and that no special diet is followed. The anastomoses in this group were almost all between 3 and 5 cm. above the dentate line. Colonic studies such as those of Gaston have been carried out here in many of these cases with the expected finding that liquid introduced into the bowel was controlled in a normal fashion.

Six of the 33 patients have failed to resume their normal bowel habits, but have some degree of control. In some cases the present difficulties clearly have to do with a persisting tendency to stenosis. It is anticipated that the symptoms will disappear as the tendency to stenosis becomes less. In other cases, the patients have apparently, against advice, become habituated to irrigations and physics. They

15. Goligher, J. C.: The Functional Results After Sphincter-Saving Resection of the Rectum, Ann. Roy. Coll. Surgeons England **8**:421-439 (June) 1951.

have some control, the degree of which cannot be adequately evaluated, but the status of the bowel is far from normal. The remaining patient of the group of seven without essentially normal control has definitely inadequate warning of an impending movement, and so soils himself occasionally. His control of liquid and gas is inadequate, while that for formed stool is fair. The anastomosis is oblique and lies above the dentate margin at a distance which varies from 1.5 cm. posteriorly to 3 cm. anteriorly. The case is thus very similar to one of Gaston's cases in that there is simply too little of the rectum remaining for a satisfactory rectal reflex.

INDICATIONS

Procedures such as endorectal resection are indicated chiefly for the management of lesions of the mid and upper portions of the rectum. In 34 of the 39 cases of carcinoma in the present series the lesions were situated between 6 and 10 cm. above the dentate margin. In two cases the lesions were situated more than 10 cm. above the dentate margin; in one of these the pull-through procedure was carried out, rather than segmental resection, because of inspissated stool above an obstructing lesion; in the other case the patient was seen early in the series and would probably be treated at present by segmental resection and primary anastomosis. The operation is not particularly suited for the treatment of lesions high in the rectum or in the rectosigmoid because of the excessive length of bowel necessary in such cases. Fortunately, lesions at this level are sufficiently high to permit segmental resection and primary anastomosis (low anterior resection). However, the lower the lesion in the rectum, the more difficult the anastomosis from above becomes until, at some level, depending mainly on the type of pelvis and the degree of obesity of the patient, anastomosis by suturing cannot be satisfactorily carried out. This, then, can be considered the upper level at which the endorectal resection is indicated. While there is great individual variation, the distal edge of the lesion in such cases is usually not more than 10 cm. above the dentate line.

Because lesions of the lower portion of the rectum may spread laterally as well as proximally both by direct extension and embolically through lymphatics, the zone of lateral spread must be removed in the radical treatment of carcinomas at this level. While studies of lateral lymphatic spread are inconclusive, essentially because so little tissue is available in surgical specimens for study, there is general agreement that such spread occurs primarily with lesions at the level of the levators.[16] Waugh and Kirklin [17] pointed out that lateral spread and inadequate removal of the tissues of the lateral zone of spread probably account for the extremely poor results after combined abdominoperineal resections for lesions low in the rectum. A surgical procedure such as the radical posterior resection of Dixon could well be considered for the treatment of lesions at this level.

16. (*a*) Coller, F. A.; Kay, E. B., and MacIntyre, R. S.: Regional Lymphatic Metastasis of Carcinoma of the Rectum, Surgery **8**:294-311 (Aug.) 1940. (*b*) Gilchrist, R. K., and David, V. C.: Lymphatic Spread of Carcinoma of the Rectum, Ann. Surg. **108**:621-642 (Oct.) 1938. (*c*) Grinnell, R. S.: Lymphatic Metastases of Carcinoma of the Colon and Rectum, ibid. **131**:494-506 (April) 1950.

17. Waugh, J. M., and Kirklin, J. W.: The Importance of the Level of the Lesion in the Prognosis and Treatment of Carcinoma of the Rectum and Low Sigmoid Colon, Ann. Surg. **129**:22-33 (Jan.) 1949.

Lateral spread becomes increasingly rare, judging from available studies of lateral spread, the higher the lesion is situated above the dentate margin. Grinnell [16c] in 1950 found lateral spread only with lesions within 4 cm. of the dentate margin. Similar findings have been reported by Gilchrist and David [16b] and by Coller, Kay, and MacIntyre.[16a] Lateral spread probably does not occur with lesions above the 4-cm. level unless proximal lymphatics are blocked. I agree with Bacon [13] that it is safe to consider preservation of continence if the lesion is as high as 6 cm. above the dentate margin.

The many studies of retrograde lymphatic spread of carcinomas at this level of the bowel, beginning with the work of McVay [18] some 30 years ago and continuing to the present,[19] have established the fact that retrograde spread does not occur until proximal lymphatics have been blocked, and that, in any case, such spread is never extensive. Involvement of nodes 2 cm. or more below the distal edge of the lesion has been found in less than 1% of cases studied and never unless proximal lymphatics were extensively blocked.[20] As Grinnell [21] in 1942 pointed out, in such advanced cases the lesion is probably already inoperable because of extension along the main lymphatic route upward.

The findings suggest that limited resection of the retrograde zone of lymphatic spread is probably adequate. Similarly, intramural spread is never extensive except in the case of anaplastic lesions. In any case, the sacrifice of 5 cm. of normal bowel distal to the lesion should provide an adequate margin of safety. I confess that with superficial polypoid lesions, particularly with no gross evidence of nodal involvement, I am willing to sacrifice less bowel distally if by this means a colonic stoma or incontinence can be avoided.

In actual practice, the amount of tissue removed by combined abdominoendorectal resection is identical with that removed by combined abdominoperineal resection proximally and laterally, above 3 cm. from the dentate margin. The decision to continue with the endorectal procedure is never finally made until the rectum and rectosigmoid have been freed from the hollow of the sacrum and until the superior hemorrhoidal or inferior mesenteric vessels have been divided. Consideration is taken of the several factors previously discussed and the decision is then made to continue the procedure as either a Miles combined or an endorectal resection.

SUMMARY AND CONCLUSIONS

A surgical procedure of the pull-through type (combined abdominoendorectal resection), in which fecal continence rather than merely a sphincter capable of contracting is preserved, has been employed in 41 cases. The essential departure from more familiar sphincter-saving procedures is that the most distal 3 to 4 cm. of rectum, as well as the sphincter and levator ani muscles, is saved intact. Rectal sensation, on which fecal continence is dependent, is thus preserved. The only

18. McVay, J. R.: Involvement of the Lymph-Nodes in Carcinoma of the Rectum, Ann. Surg. **76**:755-767 (Dec.) 1922.

19. Goligher and Hughes.[14b] Goligher.[15] Coller, Kay, and MacIntyre.[16]

20. Best, R. R.: Rectosigmoidectomy with Anastomosis in Carcinoma of Rectum and Rectosigmoid, J. Internat. Coll. Surgeons **13**:203-208 (Feb.) 1950.

21. Grinnell, R. S.: The Lymphatic and Venous Spread of Carcinoma of the Rectum, Ann. Surg. **116**:200-216 (Aug.) 1942.

serious complication inherent in such procedures is slough of the sigmoid, which occurred six times in 41 cases. The slough is serious only if the gangrenous part extends above the rectal stump. This occurred in three of the six cases. While much local reaction occurred, a proximal colonic stoma was not necessary in any of the three cases. Union between the sigmoid and the rectal stump was satisfactory in all cases of carcinoma. In two cases of megacolon in the adult the union was delayed; in one such case, establishment of a proximal colonic stoma became necessary.

The cut end of the sigmoid tends to become stenotic until epithelization occurs. Intermittent digital dilation is usually necessary for approximately three months after operation. The procedure is indicated for the treatment of lesions of the mid and upper portions of the rectum or of lesions approximately 6 to 10 cm. above the dentate line. Lesions above this level can usually be better managed by segmental resection with primary anastomosis, and lesions below this level should not be managed by endorectal resection, since in this procedure the zone of lateral lymphatic spread is not removed.

TREATMENT OF HEMANGIOMAS

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FROM THE care and study of a large group of patients with hemangiomas of wide variety in location, duration, former treatment, pathology, requirements for treatment and presenting of many problems of repair of destructive processes, surgical removal, and radiation lesions, observations thought to be of importance are outlined.

Bright-red arterial hemangiomas require careful and prompt treatment in most instances. These bright-red growths occur early in infancy and are possibly true neoplasms, whereas purely congenital types, such as port-wine stains and the bluish cavernous lesions, are possibly anomalies of formation of the skin, or the vessels, or both. These usually do not take on neoplastic tendencies, except occasionally later in life.

These bright-red growths usually start as small areas and may increase rapidly in size, so that a whole feature of the face may be destroyed while some plan of "wait and see" is being followed or hope of retrogression is held.

There are many reports of impressions that regression does occur that are often quoted, as, for example, that of John Duncan,[1] who wrote in 1888 that "more than half the total of nevi of mixed and subcutaneous type automatically disappear." No pathological microscopic study or photographs have been included in reports of any sizable series of cases to support the policy of withholding treatment and waiting for spontaneous recession. Even if this did occur, however, a reason for not waiting for such recession when features are involved is that the feature itself may be destroyed in the process of growth during the time required for the natural decision as to the destiny of the tumor.

The important point is that if these bright-red arterial hemangiomas could be recognized as dangerous when they first occur as small lesions, they could be dealt with easily by simple excision or cautery removal, or by minimal interstitial radiation therapy with gold radon seed of 0.1-mc. content or less, as has been reported.[2]

From the Department of Surgery, Washington University School of Medicine.

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

1. Duncan, J.: On the Value of Electrolysis in Angioma and Goitre, Brit. M. J. **2**:984-986 (Nov. 3) 1888.

2. Brown, J. B., and Fryer, M. P.: Hemangiomas: Treatment and Repair of Defects, Surg., Gynec. & Obst. **95**:33-44 (July) 1952.

When the tumor is of some size, the same procedures described above can be carried out with sometimes a staged plan of treatment: (1) control of the growth with interstitial gold radon seed, and (2) secondary surgical excision and repair, as indicated, of any remaining scarred surface or deformity.

In repeated instances it has been possible to save from destruction the eyelid, nose, lip, ear, or auditory canal by the careful use of interstitial radiation in doses of 0.1 mc. or less of radon in gold seed per 1 cc. of tissue irradiated. With use of

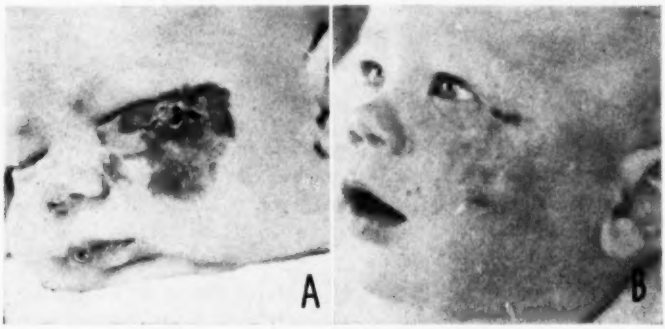

Fig. 1.—*A*, rapidly growing arterial hemangioma of face and eyelid starting from a small area, as is usually seen, although it may be present at birth. *B*, result of use of interstitial radon in 0.1-mc. gold seeds for each cubic centimeter of tissue to be treated, the eyelid having been saved from destruction. There is complete control of activity of the growth, but the atrophic skin has settled down and can be replaced if needed by multiple-stage excision or resection and free grafting from the region of the ear, neck, or below the breast.

Fig. 2.—Widespread arterial hemangioma on surface and deep, controlled with interstitial gold radon seeds of 0.1-mc. content. Skin of atrophic character remains and has settled down over tumor site. Because of involvement of eyelids and orbit and of possible late change, the area is removed and repaired with a full-thickness graft from behind the ear.

local anesthesia much of the work can be done as an outpatient procedure by scheduling the patient's arrival at the time specified for the proper radon content of the gold seed. Instances of such use are shown in Figures 1 and 2.

This small unit dose may have to be repeated, but it is best to realize and to inform the patient or his parents that any radiation dose may leave scarred, atrophic surface skin after control of the tumor, when the already damaged skin settles down in place, so that secondary surgical repair is frequently necessary (Fig. 2). The

same is true of the growth and development of bone, cartilage, and soft tissue, except that this minimal radiation dose is considered the least likely to cause secondary changes, as seen in typical radiation burns.

The type of radiation is not important, but the dose should be kept low. External x-ray treatment may be used if the baby can be held quiet enough for it, or other, more recent forms of delivering the required dose, such as other removable subcutaneous implantations of induced radioactive material may be employed.

It may be repeated that the dose advocated here is so low that it may seem at variance with other recommended doses of 0.25 to 0.5 mc., but this higher dose is too likely to result in late changes to warrant its use in treatment of these tumors. This is not true of other, more destructive or dangerous tumors. The main hope in

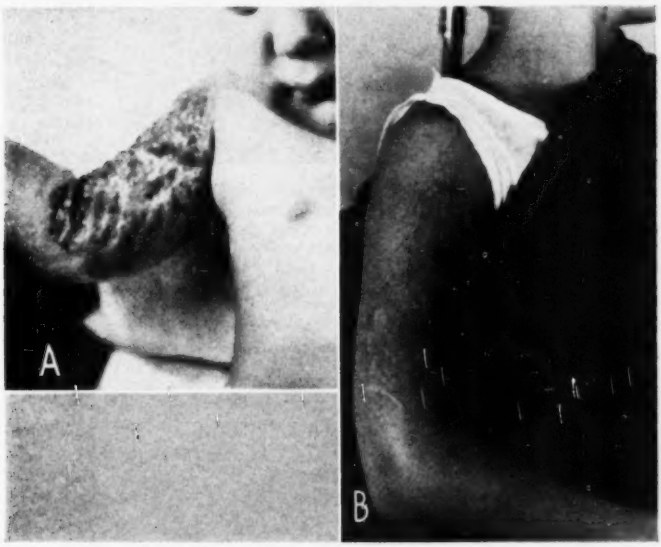

Fig. 3.—*A*, intractable hemangioma growing on arm with open area, result of multiple treatments (elsewhere). *B*, complete excision of the area and repair with free split skin grafts. This type of resection is possible on flat surfaces, but when features are involved such wide resections are avoided, if possible, by use of interstitial or other forms of radiation, with late repair of scarred area as indicated.

cases of these neoplasms is to control the tumor but leave the supporting tissue intact, with the chance for the most normal development.

Intractable growths, especially on flat surfaces, may require wide, deep excision and immediate or secondary skin grafting, as illustrated in Figure 3.

Port-wine stains or capillary hemangiomas in exposed surfaces can often be dealt with best by using cosmetics, but men and boys seldom wish to carry out this procedure, and if the lesion on the face is objectionable enough, there is little to offer except excision and grafting. Abrasive or freezing methods may occasionally suffice but are apt to result in substitution of one deformity for another. Pigment injection (tattoo) is not sufficiently reliable to be advocated at present. This method

was first reported from the surgical service at Valley Forge Hospital, but its value has not been substantiated in a large series of satisfactory cases observed over a long period.

Fig. 4.—*A*, scar of old hemangioma controlled with radiation. No hemangioma is left, but there remains a chronic progressive radiation lesion that needs to be resected and the defect repaired. *B*, complete resection of the skin has been done and resurfacing with free grafts carried out. The upper lip is resurfaced with a graft from above the clavicle, as giving the best function and color match.

Fig. 5.—Type of growth suitable for at least trial use of multiple-suture procedure in an endeavor to obliterate the lesion but leave the surrounding tissues intact—in this instance the cheek and adjacent features. Multiple procedures may be required.

The port-wine stain is not subject to relief by irradiation, and some of the worst burns that have been seen requiring repair have come from the treatment of such lesions with radiation. This is illustrated in Figure 4.

Cavernous hemangiomas, which are areas of large channels containing mainly blood on the venous side of the circulation, seem to be anomalies at first but later in life may take on extension and enlargement and actually endanger life from position or injury.

These growths do not respond to radiation very well, but partial control may be secured from the small interstitial dose. They may also be excised if situated so that this may be carried out. The injection of sodium morrhuate has given some excellent retrogressions, if not complete cures.

Multiple-suture obliteration of these cavernous growths has been used to good advantage for several years, but, owing to limited observation, has not been reported. The effort in this procedure, again, is to control the lesion but to leave the supporting structures or features intact. The intractable cavernous tumor illustrated in Figure 5 is one in which this method is applicable. The procedure consists of introducing chromic absorbable surgical (gut) sutures directly through the surface into the tumor and out and tying them on the surface, the attempt being made to avoid sloughing from too many sutures. The procedure can be repeated or combined with others, such as injection or resection.

Lymphangiomas or hygromas are mentioned as being somewhat related and as possibly having venous connections or involvements. They are not neoplastic, except for enlargement, deforming areas, and contour anatomy. They are dealt with by excision, injection, packing, control of venous connections, and multiple-suture procedures.

In all these growths the microscopic picture bears little relation to the seriousness of the clinical picture. The most benign-appearing lesion can be malignant by position or involvement or in relation to the excessive difficulty of care and repair. Regarding malignant tendencies, these growths can cause death, which is the true meaning of malignancy, without appearing malignant microscopically. This is another instance in which the clinical picture is of greater importance than the microscopic findings.

Solid endotheliomas, or hemangioendotheliomas, are of a more neoplastic nature and may require microscopic study for final diagnosis. Their treatment is about the same. Surgical excision is used when possible, but interstitial radiation is relied on when features are to be saved, and it is usually employed in the parotid region in children in an attempt to preserve the seventh cranial nerve. Somewhat higher dosages may be required in treating these growths.

Plastic surgery repair of the defects of these lesions is frequently necessary, either as a primary procedure following removal or for repair of later scarring and loss of contour anatomy or radiation lesions (Figs. 2, 3, and 4).

OBSERVATIONS ON ETIOLOGY OF ESOPHAGEAL VARICES

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THE ENCOURAGING results of recent operations designed to relieve portal hypertension have brought renewed interest in the nature of varices of the esophagus. It is generally assumed that these lesions, often fatal because of massive bleeding from them, are secondary to increased pressure within the portal system of veins. In the great majority of patients, this is true. Occasionally, however, esophageal varices are found in the absence of portal hypertension, and for these persons operations designed to correct increased portal venous pressure would be ill-advised. The problem is illustrated by a patient recently studied at City Hospital, Cleveland.

REPORT OF A CASE

J. B., a 42-year-old white man, was admitted to City Hospital on Jan. 15, 1951, because of severe frostbite of both feet following six days of exposure to freezing weather. The patient had been in good health in the past but admitted a poor dietary intake and chronic alcoholism. Examination on admission revealed superficial gangrene of the left foot with cyanosis and diminished skin temperature of the right foot. Heparin was included in the treatment for frostbite, but its administration was discontinued before the end of the second day because gangrene had become well demarcated. On the third hospital day the clotting time of the blood was normal.

Six days after admission the patient had massive hematemesis and melena. The hematocrit value dropped to 22%, and the red blood cell count to 2,300,000 per cubic millimeter. Bleeding and clotting times were normal. Two thousand cubic centimeters of whole blood was given. Results of physical examination at that time were not remarkable except for the local findings in the extremities; the spleen was not palpable. Liver function tests on the 8th hospital day revealed normal values for the total proteins, albumin-globulin ratio, cephalin-flocculation, and thymol turbidity. The blood cholesterol was 140 mg. per 100 cc., and the cholesterol esters were 52 mg. On the 11th hospital day barium studies of the upper gastrointestinal tract revealed esophageal varices but no other abnormalities (Fig. 1). There was no further hemorrhage. Esophagoscopy two days later revealed the presence of many large varices in the lower esophagus. The patient did not bleed again, and on the 44th hospital day a transmetatarsal amputation was done through the left foot, with subsequent normal healing. On the 50th hospital day results of liver function tests, including that of sulfobromophthalein (bromsulfalein) sodium excretion, were normal. In view of the patient's long addiction to alcoholism, a diagnosis of portal hypertension with esophageal varices secondary to Laennec's cirrhosis was made.

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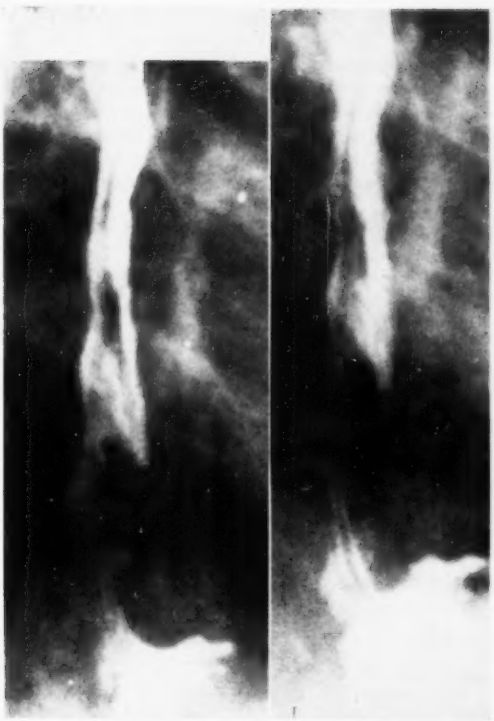


Fig. 1.—Roentgenogram showing evidence of esophageal varices preoperatively.

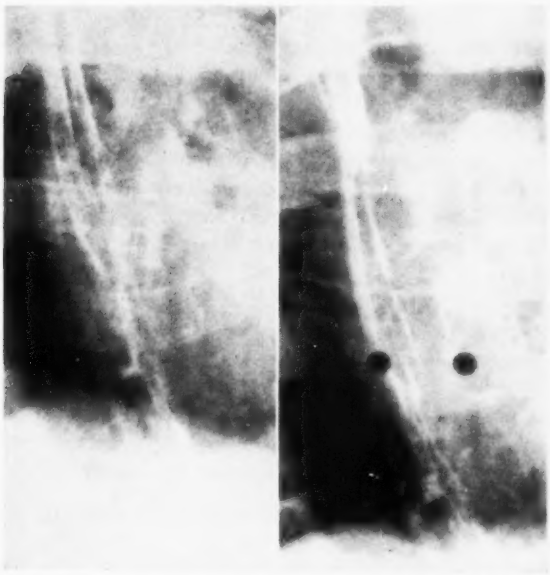


Fig. 2.—Roentgenogram taken six months after exploration showing no evidence of esophageal varices.

It was elected to do a portacaval anastomosis, and operation was done on the 51st hospital day. At exploration no abnormality was found within the abdomen. The liver, spleen, and stomach were grossly normal. The pressure in the portal vein was 13 cm. of isotonic sodium chloride solution and in the gastroepiploic veins 15 cm. There was no evidence of abnormally developed collateral circulation. Since these findings failed to corroborate the diagnosis of portal hypertension, the contemplated portacaval shunt was abandoned. A specimen of the liver was taken for biopsy.

The patient made an uneventful recovery and was discharged nine days after operation. Examination of the liver specimen showed only a very slight fatty metamorphosis. The patient has remained asymptomatic to the present time. Esophagograms on July 16, 1951, were normal (Fig. 2).

This case stimulated a review of all autopsy protocols at City Hospital in which a diagnosis of esophageal varices had been made. Thirty-one protocols were reviewed. In 22 cases (Table 1) Laennec's cirrhosis and other changes characteristic of portal hypertension, namely thickening of the portal vein and abnormal collaterals between the portal and caval systems, were observed. In nine cases esophageal varices were found in the absence of Laennec's cirrhosis (Table 2).

TABLE 1.—*Major Associated Diseases Found in Thirty-One Cases of Esophageal Varices Proved at Autopsy*

Major Disease Process	No. of Cases
1. Laennec's cirrhosis (with associated organic heart disease, 6 cases; without organic heart disease, 16 cases)	22
2. Hypertensive cardiovascular disease	3
3. Coronary artery heart disease	2
4. Syphilitic heart disease	1
5. Rheumatic heart disease	1
6. Bronchogenic carcinoma with metastases	1
7. Chronic cholecystitis and cholelithiasis	1

In three of these nine cases, esophageal varices were associated with other abnormalities of the liver but without evidence of portal hypertension: in Case 4, cardiac cirrhosis; in Case 7, focal acute necrosis and minimal periportal fibrosis, and in Case 8, small metastatic nodules secondary to bronchogenic carcinoma. Esophageal varices were found associated with cardiac hypertrophy and dilatation and chronic passive hyperemia of the liver without extrahepatic portal block in five of the nine cases. In the ninth case esophageal varices were found with a normal liver and spleen and with no evidence of a block anywhere in the portal system of veins.

COMMENT

The commonest cause of esophageal varices is portal hypertension, and in most instances this is due to Laennec's cirrhosis. Higgins,[1] in a review of 115 cases of varices in the esophagus, found that in 80% of them the varices were associated with Laennec's cirrhosis. Other causes (Table 3) of intrahepatic block of the portal system causing portal hypertension include postnecrotic cirrhosis, toxic cirrhosis, schistosomiasis, gumma of the liver, and thrombosis of the hepatic vein.[2]

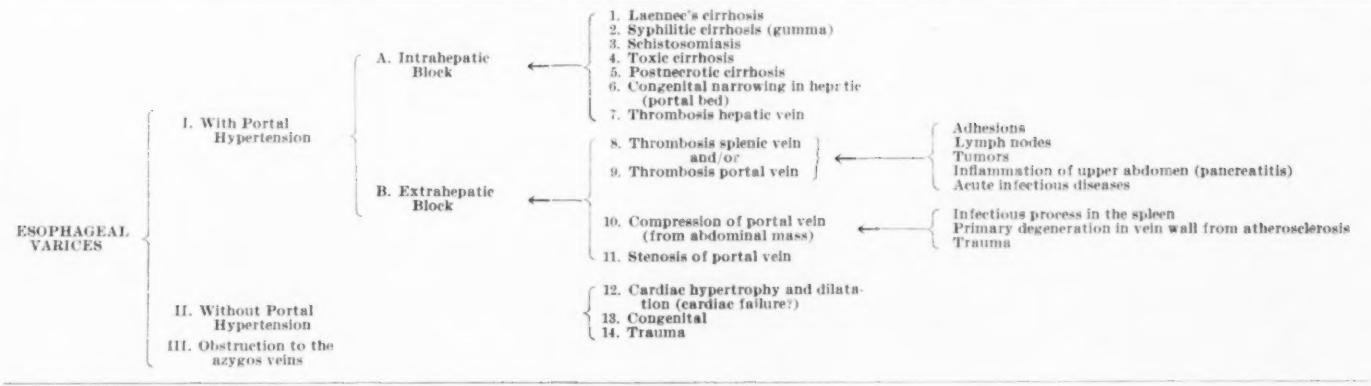
1. Higgins, W.: The Esophageal Varix: A Report of 115 Cases, Am. J. M. Sc. **214**:436, 1947.

2. Linton, R.; Hardy, L., and Volwiler, W.: Porta Caval Shunts in the Treatment of Portal Hypertension, Surg., Gynec. & Obst. **87**:129, 1948.

TABLE 2.—*Significant Findings in Nine Cases of Esophageal Varices Not Associated with Laennec's Cirrhosis*

Case No.	Age (Yr.)	Sex	Clinical History	Ante-mortem Diagnosis	Location	Major Pathology	Liver	Spleen	Heart Weight, Gm.
1	45	M	Admitted in coma with a recent hemiplegia; died after 5 days	No	Area not recorded	Recent brain infarction; recent myocardial infarction; cardiac hypertrophy	Chronic passive hyperemia; weight 2,400 gm.	Chronic passive hyperemia; weight 150 gm.	510
2	48	F	Hypertension with cardiac failure; died in uremia	No	Lower third	Arterial and arteriolar nephrosclerosis; cardiac hypertrophy	Normal; weight 1,450 gm.	Normal; weight 110 gm.	615
3	56	M	Admitted with a recent cerebral vascular accident; died 3 days later	No	Cardioesophageal junction	Arterial and arteriolar nephrosclerosis; cardiac hypertrophy	Chronic passive hyperemia; weight 1,450 gm.	Normal; weight 175 gm.	475
4	60	M	Severe coronary artery heart disease with failure; died with hemoptysis	No	Generalized	Multiple pulmonary emboli; phlebothrombosis in popliteal vein; cardiac hypertrophy	Cardiac cirrhosis; weight 1,450 gm.	Infarct; weight 200 gm.	550
5	56	M	Admitted in cardiac failure; died after a gradual downhill course	No	Area not recorded	Syphilitic aortitis; aortic aneurysm; cardiac hypertrophy	Chronic passive hyperemia; weight 1,025 gm.	Chronic passive hyperemia; weight 90 gm.	550
6	58	M	Admitted in cardiac failure; died on the 9th hospital day	No	Lower third	Rheumatic heart disease; cardiac hypertrophy	Chronic passive hyperemia; weight 1,510 gm.	Chronic passive hyperemia; weight 150 gm.	575
7	65	M	Admitted because of hematemesis; died on the 9th day	Yes	Cardioesophageal junction	Ruptured esophageal varices; cardiac hypertrophy	Focal acute necrosis; periportal fibrosis (minimal); weight 1,750 gm.	Chronic passive hyperemia; weight 650 gm.	550
8	57	M	Admitted for terminal care with bronchogenic carcinoma and metastases; died after 7 weeks	No	Cardioesophageal junction	Bronchogenic carcinoma with widespread metastases	Small metastatic nodules; weight 4,200 gm.	Chronic passive hyperemia; weight 150 gm.	270
9	51	F	Admitted because of hemoptysis; died within 24 hours	No	Lower third	Ruptured esophageal varix; chronic cholecystitis with cholelithiasis	Chronic passive hyperemia; weight 1,100 gm.	Acute hyperplasia; weight 275 gm.	325

TABLE 3.—*Conditions Associated with Esophageal Varices*



Extrahepatic block of the portal venous system likewise causes portal hypertension and produces esophageal varices. The suggestion has been made, too, that obstruction of the azygos system may cause esophageal varices.[3]

It is not appreciated generally that esophageal varices may be found in the absence of disease of the liver or portal system. Nochimowski [4] in 1932 reported two cases in which death was caused by a hemorrhage from ruptured esophageal varices, and in both the liver was entirely normal. No cause for the development of varices could be found. He collected eight similar cases from the literature. Among the total of 10 patients, 7 were adults, 2 were children, and 1 was an infant, 3 days of age.

Friedman [5] in 1934 reported the case of a 3½-year-old boy who had had four episodes of severe hematemesis after he had reached the age of 18 months. The child died of hemorrhage eight days after hospitalization, and postmortem examination revealed no abnormalities other than extremely large venous plexuses in the lower part of the esophagus. Lichtman [6] in 1942 stated that in rare instances esophageal varices have been found in cases of chronic alcoholism without cirrhosis of the liver. The phlebectasia has been attributed to the caustic action of alcohol on the mucosa of the esophagus. In 1947 Bell [7] stated that in rare instances of fatal hemorrhage from esophageal varices there is no obstruction in the portal circulation and no enlargement of the spleen.

In 1949 Weinberg,[8] reviewing 1,189 consecutive autopsies, found 95 cases of esophageal varices. In 18 cases of this group the varices were associated with Laennec's cirrhosis, 2 with biliary cirrhosis, and 1 with toxic cirrhosis. Presumably all these patients had had portal hypertension during life. In 49 cases, 52% of the entire group, esophageal varices were associated with coronary artery heart disease without evidence of cirrhosis or portal hypertension. In 20% of this series, varices were associated with other miscellaneous conditions, none of which are commonly associated with portal hypertension.

The case reported here is of interest because it illustrates the presence of esophageal varices from which massive hemorrhage had occurred in the presence of normal portal vein pressures. Gray and Whitesell [3] imply that they have had a similar case. There can be little doubt of the existence of esophageal varices, since the roentgenographic findings were clear and were corroborated by direct visualization through the esophagoscope. The failure to demonstrate the varices by roentgenography after a five-month interval suggests that the condition was a reversible process. In the cases studied at autopsy there is little reason to doubt the existence of portal hypertension in the 22 cases with the diagnosis of Laennec's cirrhosis.

3. Gray, H. K., and Whitesell, F. B., Jr.: Hemorrhage from Esophageal Varices: Surgical Management, Ann. Surg. **132**:798, 1950.

4. Nochimowski, J.: Fatal Hemorrhages from Varices of Esophagus in Cases Without Cirrhosis of the Liver, Ztschr. Path. **43**:463, 1932; abstracted, J. A. M. A. **99**:1643, 1932.

5. Friedman, E.: Esophageal Varix: Report of Case in a 3½ Year Old Child, Not Dependent on Liver Cirrhosis, J. Pediat. **4**:641, 1934.

6. Lichtman, S.: Diseases of the Liver, Gall Bladder and Bile Ducts, Philadelphia, Lea & Febiger, 1942.

7. A Textbook of Pathology, edited by E. Bell, Philadelphia, Lea & Febiger, 1947.

8. Weinberg, T.: Observations on the Occurrence of Varices of the Esophagus in Routine Autopsy Material, Am. J. Clin. Path. **19**:554, 1949.

In the cases with cardiac cirrhosis, focal acute necrosis with minimal periportal fibrosis, and the small metastatic nodules, portal hypertension might have been present but this is unlikely; it is especially unlikely in the case with small metastatic nodules, since even with massive liver replacement by metastasis portal hypertension is rare. In Weinberg's series [8] there were five cases with bronchogenic carcinoma and mediastinal masses associated with esophageal varices. These suggest obstruction of the azygos vein as the cause for the varices.

In the cases associated with cardiac hypertrophy and dilatation and chronic passive hyperemia of the liver, the findings suggest strongly the possibility of "suprahepatic" venous obstruction as mentioned by Welch.[9] He states: "Although this mechanism of suprahepatic venous obstruction produces some increase in pressure in the portal system the increase is never so significant that it requires treatment in these states, since esophageal varices do not develop nor is massive gastrointestinal hemorrhage observed in patients with cardiac cirrhosis." Increased intravenous pressures in the subdiaphragmatic inferior vena cava have been found in subjects with congestive heart failure.[10] Normal persons have an average pressure of 134 mm. of water, while the pressure of those in failure averages 267 mm. It is reasonable to assume that an abnormally elevated vena caval pressure coexists with cardiac hypertrophy, and this increased pressure is transmitted to the hepatic bed. Our findings suggest that esophageal varices are capable of development in these circumstances.

Case 9 is of interest because death was due to rupture of an esophageal varix and no cause of the existence of varices could be established, the protocol reading: "There are no anatomical findings present to account for the esophageal varices in this case." This case is similar to those reported by Nochimowski and Friedman. It is possible that the patient on whom we performed an exploratory operation might also fit into this group.

The cause of esophageal varices in the presence of portal hypertension is assumed to be the increased flow of blood through the capillary-like anastomosis between the portal and caval systems at the gastroesophageal junction. These veins are poorly supported by loose connective tissue and are not able to withstand the increased intravenous pressure and dilatation which develop. In addition to this factor, some authors [11] have postulated that, since esophageal varices are intrathoracic in location, during inspiration the negative pressure in the thorax is transmitted to the varices while the intra-abdominal pressure increases, causing a rise in portal pressure and thereby facilitating dilatation of the esophageal veins. The absence of coronary vein valves has also been cited as a contributory factor to the easy regurgitation of blood from the portal to the caval system.[1]

In the absence of portal hypertension the cause of esophageal varices is conjectural. Weinberg [8] postulated that in these circumstances esophageal varices might develop in foci of congenital weakness of the venous channels of the esophagus. Higgins [1] suggests that trauma to the esophagus results in varices. But neither of these two ideas has been proved.

9. Welch, C.: Portal Hypertension, New England J. Med. **243:** 598, 1950.

10. Maxwell, M. H.; Breed, E., and Schwartz, I. L.: Renal Venous Pressure in Chronic Congestive Heart Failure, J. Clin. Invest. **29:**342, 1950.

11. Higgins,[1] Lichtman.[6]

In our series, five patients with esophageal varices had cardiac hypertrophy and dilatation. This suggests that some mechanism exists which produces abnormally elevated inferior vena caval pressures. This caval hypertension is transmitted secondarily through the liver to the portal system and may cause esophageal varices. In the case presented in this report there was no apparent cause for the varices.

SUMMARY

A case is reported in which massive hematemesis from esophageal varices, proved by roentgenography and esophagoscopy, was found, and in which at operation portal vein pressures were found to be normal.

A study was made of 3 autopsy protocols in which a diagnosis of esophageal varices was made. The following associated conditions were found: (1) In 22 cases, esophageal varices were associated with Laennec's cirrhosis and portal hypertension; (2) in 3 cases, varices were associated with other abnormalities of the liver not usually producing portal hypertension; (3) in 5 cases, varicosities were associated with cardiac hypertrophy and dilatation and chronic passive hyperemia of the liver; (4) in one case, varices were found and no cause for their existence could be established.

HYPERTENSION AND ITS RELATION TO HEPATIC CIRCULATION

Effects on Arterial Pressure of Variations in the Blood Supply of the Liver in Normotensive, Hypertensive, and Cirrhotic Dogs, and in Hypertensive Patients with Portal Cirrhosis

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ONE OF us (J. K. B.) previously reported that changes in systemic blood pressure do not occur when hepatic vascular moieties are altered in the experimental animal or in man.[1] This fact and the observation that portal cirrhosis is rarely associated with arterial hypertension prompted us to study the relationship of liver circulation to changes in systemic blood pressure.

The results of vagaries in hepatic arterial, portal, and hepatic venous flow on systemic blood pressure were measured in angiotonin units and correlated with manometric determinations. Combinations of venous and arterial alterations were also produced, and the corrective effects of such maneuvers, one against the other, were recorded. Three groups of dogs were studied: (*a*) normotensive, (*b*) hypertensive, and (*c*) cirrhotic. Two cases of Laennec's cirrhosis and ascites associated with arterial hypertension are also included in this report.

METHODS AND MATERIALS

Healthy mongrel dogs were made hypertensive by unilateral nephrectomy with, at the same time, enfoldment of the opposite kidney in a silk envelope, which had been dusted with sulfathiazole before its application. Blood pressures were recorded by intra-arterial manometric readings and if a constant level of hypertension was not produced in six months to a year, the Goldblatt clamp was applied to the renal artery. Each week a record was made of the dogs' weight, blood pressure, hematocrit, blood urea nitrogen, and angiotonin units. The assay of renin substrate in the plasma was tested by the method described by Helmer and Griffith[2]: One milliliter of heparinized plasma was mixed with 3 ml. of angiotonase-free hog renin, equivalent to 3 Goldblatt units, and incubated for five minutes in a water bath at 37 C. At the

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From the Departments of Surgery, the Indianapolis General Hospital and the Indiana University Medical Center.

Dr. Kenneth Kohlstaedt, Dr. Oscar Helmer and Dr. Robert Shipley, of the Lilly Research Laboratories, gave valuable aid in this study.

1. Berman, J. K.; Koenig, H., and Muller, L. P.: Ligation of Hepatic and Splenic Arteries in Treatment of Portal Hypertension: Ligation in Atrophic Cirrhosis of the Liver, A. M. A. Arch. Surg. **63**:379-389 (Sept.) 1951.

2. Helmer, O. H., and Griffith, R. S.: Biological Activity of Steroids as Determined by Assay of Renin-Substrate (Hypertensinogen), Endocrinology **49**:154-161 (Aug.) 1951.

end of the incubation, dilute hydrochloric acid was added to adjust the pH to 5.1 or 5.2; the mixture was then placed in a boiling water bath for 10 minutes. The coagulated protein was removed by centrifugation and the clear supernatant fluid poured off and tested for angiotonin by intravenous injection in pithed cats, prepared as described by Shipley, Helmer, and Kohlstaedt.[3] The various samples of supernatant fluid were compared with a lyophilized laboratory standard of angiotonin, 30 γ of which is designated as 1 unit. The quantity of renin substrate found in the plasma is expressed as units of angiotonin formed by 1 ml. of plasma.

The dogs were made cirrhotic by the application of a polyethylene band around the inferior vena cava, just above the diaphragm, constricting the lumen of this vessel to about 50% of its circumference.[4] In three to six weeks ascites begins to form, and morphologic changes may be demonstrated in the liver. Other dogs were made cirrhotic by preliminary partial ligation of the

Fig. 1 (Dog B 83).—Photomicrograph of liver taken at autopsy. Death occurred three hours after injection of 10 cc. of 0.9% dicetyl phosphate in olive oil into the portal vein, which had not been previously constricted. There is extensive hemorrhagic necrosis. Hematoxylin and eosin; stain; × 600.

portal vein and then, three to six weeks later, the injection of 10 cc. of a 0.9% solution of dicetyl phosphate in olive oil into the portal vein or the injection of 5 cc. of a 0.9% dicetyl phosphate in olive oil into the distal end of the previously tied hepatic artery. Cirrhosis was also induced by injecting 5 cc. of a 2% aqueous solution of sodium dicetyl phosphate into the portal vein three weeks after preliminary partial ligation of the portal vein. It was found in previous

3. Shipley, R. E.; Helmer, O. M., and Kohlstaedt, K. G.: The Presence in Blood of a Principle Which Elicits a Sustained Pressor Response in Nephrectomized Animals, Am. J. Physiol. **149**:708-720 (June) 1947.

4. Berman, J. K., and Hull, J. E.: Experimental Ascites: Its Production and Control, Surg. **32**:67-75 (July) 1952.

experiments [5] that if the portal vein were partially constricted three to six weeks prior to the injection of dicetyl phosphate in olive oil the animal could tolerate up to 10 cc. doses of this material without fatality. If this preliminary constriction were not induced, then 5 cc. of the oil solution injected in the portal vein was lethal. The reaction to dicetyl phosphate is that of a foreign-body irritant, but the result is not unlike "cardiac" cirrhosis with early foreign-body fibrosis (Fig. 1). When injections were made into the hepatic artery without preliminary ligation of this vessel at its source, death followed within a few hours owing to acute hepatic insufficiency. If the artery was tied and then dicetyl phosphate injected, three to six weeks later, the animals survived. We concluded that slow constriction of the portal vein stimulates the development of intrahepatic venous tributaries which tolerate larger amounts of foreign material probably due to an increase in the amount of collateral circulation within the liver

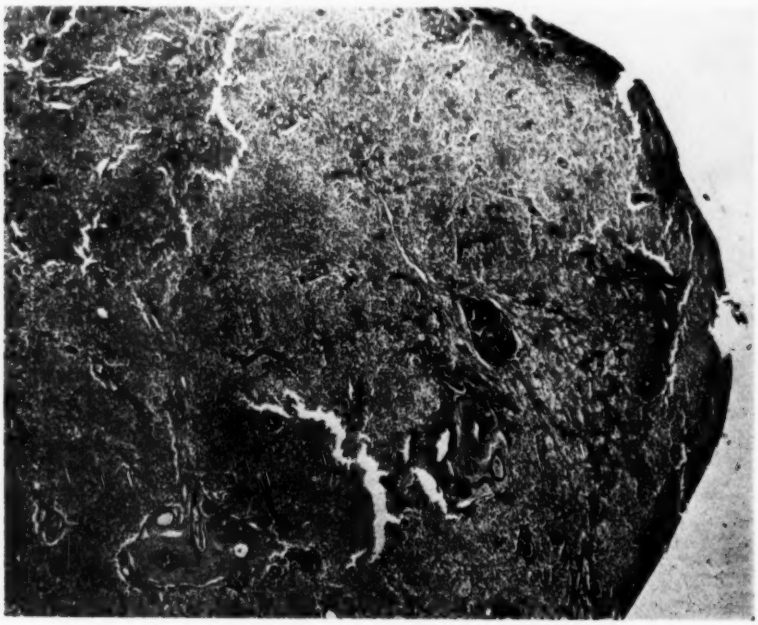

Fig. 2 (Dog B 77).—Liver biopsy specimen obtained seven days after injection of 10 cc. of 0.9% dicetyl phosphate in olive oil into portal vein, which had been constricted three weeks previously. Note fatty infiltration and early foreign-body reaction giving rise to scattered granulomas. Hematoxylin and eosin stain; × 32.

(Fig. 2). Preliminary ligation of the hepatic artery permits collateral circulation to form preventing hepatic necrosis.

ROLE OF THE LIVER IN SYSTEMIC HYPERTENSION

The staff of the Indianapolis General Hospital Hypertension Clinic made a careful study of the incidence of portal cirrhosis in patients with high blood pressure.[6] They found that Laennec's cirrhosis occurs in less than 3% of hyper-

5. Berman, J. K.; Koenig, H., and Saint, W. K.: Ligation of the Hepatic and Splenic Arteries in the Treatment of Portal Hypertension, Indiana Univ. Quart. Bull. **12**:99 (Oct.) 1950.

6. Levi, L.: Personal communication to the authors.

tensives. Ratnoff and Patek [7] found combined cardiovascular renal disease in about 6% of their 386 cases of Laennec's cirrhosis. The chief complications of cirrhosis in their series were intercurrent infections, anemia, abdominal hernia, peptic ulcer, and portal vein thrombosis; the chief causes of death were cholemia, hematemesis, postoperative complications, pneumonia, and intercurrent infections.

The reasons for the comparative rarity of hypertension in Laennec's cirrhosis are not definitely known. However, many observers have concluded that the liver plays an important role in the normal regulation of blood pressure. If this hypothesis is true, then hepatic impairment would interfere with mechanisms ordinarily invoked in the production of hypertension.

The two more prominent theories of hepatic pressure regulation are based on the concept that the liver produces or fails to produce certain hormones or chemical substances which stimulate or inhibit the contraction of arterioles, or that the liver and portal bed act as a tremendous expansible reservoir which progressively reduces blood volume by the storage of large quantities of blood and the loss of great amounts of the blood volume through plasmapheresis as ascites.

Hepatogenic and nephrogenic substances may react to produce hypertensive activators as follows: Renin, and enzyme, is normally produced by the kidney, but this substance is elaborated in larger quantities when the kidney is ischemic or anoxemic. Renin subtrate (hypertensinogen), an $alpha_2$ globulin fraction of the plasma, is the precursor of angiotonin (hypertensin) and is formed in the liver. When renin substrate reacts with renin, it produces the vasopressor polypeptide angiotonin. A different relationship is described by Shorr, Zweifach, and Furchgott,[8] who believe the two components responsible for changes in arterial pressure are the vasodepressor material of hepatic origin (VDM) and the vasoexcitor material of renal origin (VEM). These factors combine to form a vascular homeostatic system, and the preponderance of one over the other may result in hypotension or hypertension. Many experiments have been done in an effort to prove the relationship of the liver to systemic hypertension.

The removal of the liver in hypertensive dogs causes a fall in blood pressure, and the same results may be produced by the use of hepatotoxic agents, such as chloroform or carbon tetrachloride. The latter results are explained on the basis of liver damage which, in turn, reduces the amount of substrate produced by this organ. Davis and Tanturi [9] obtained a fall in blood pressure after partially occluding the portal vein and the hepatic artery in dogs. They believe that either the damaged liver is unable to manufacture renin substrate, which is an essential part of the pressor reaction of renal hypertension, or that the anoxemic liver produces active vasodepressor material, such as that demonstrated by Shorr and his co-workers, which neutralizes the vasoexcitor material elaborated by the anoxemic kidney. Perhaps both mechanisms are active.

7. Ratnoff, O. D., and Patek, A. J., Jr.: The Natural History of Laennec's Cirrhosis of the Liver: An Analysis of 386 Cases, Medicine **21**:207-268 (Sept.) 1942.

8. Shorr, E.; Zweifach, B. W.; Furchgott, R. F., and Baez, S.: Conference on Factors Regulating Blood Pressure: First to Third Conferences, 1947-1949, New York, Josiah Macy, Jr., Foundation, 1948.

9. Davis, L., and Tanturi, Carlos, A.: Liver as a Factor in Experimental Renal Hypertension, A. M. A. Arch. Surg. **62**:325-3334 (March) 1951.

Experiments which we have done within the last four years seem to show that portal pressure is not inversely related to systemic blood pressure if long term studies are done. The immediate relationship between a sudden rise in portal pressure and arterial pressure has recently been studied by Peck and Grover.[10] These investigators have demonstrated a portal bed-hepatic reflex which manifests itself as an acute arterial hypotension within approximately 30 seconds after portal pressure has been elevated. The reflex is probably hormonal rather than neurogenic and is followed by a more gradual decline in blood pressure until the death of the animal, which occurs within a few hours. We, too, have noted that there is a temporary inverse relationship between elevated portal pressure and systemic blood pressure in the normal animal. This is due to a transient decrease in circulating blood volume. However, in our animals with partial occlusion of the inferior vena cava, ample collateral circulation gradually develops, and, as portal pressure falls, arterial pressure rises to normal levels. In hypertensive dogs with gradually induced portal stasis (and ascites) there is a sustained fall in arterial pressure, although portal pressure is not elevated. There is no decrease in angiotonin units.

OBSERVATIONS ON EXPERIMENTAL ANIMALS

Group A—Normal Dogs

I. Hepatic Artery Ligation.—1. Ligation and Division of the Hepatic Artery in Dogs Protected with Penicillin: (*a*) Ligation at the celiac axis produced no immediate or late effects observable on the systemic blood pressure.

(*b*) Ligation of the hepatic and splenic arteries close to the celiac axis produced no changes in blood pressure.

(*c*) With ligation of the hepatic artery branches as they enter the liver, all animals died within 24 to 48 hours of liver necrosis or gangrene of the gall bladder with peritonitis. There was a fall in blood pressure prior to death. This could not be attributed to ligations but rather to the moribund state of the animals.

(*d*) Ligation of collaterals of the hepatic artery six weeks to three months after hepatic artery ligation at the celiac axis caused death in most of the animals within 48 to 72 hours due to liver necrosis. There was no change in blood pressure except a fall shortly before death.

II. Portal Vein Ligation and Occlusion.—1. Ligation of the portal vein produced death within 24 hours in all animals whether protected with penicillin or not. Blood pressure declined slightly immediately after ligation, then decreased more rapidly, and fell precipitously before death. The earlier fall in blood pressure was thought to be due to the entrapment of a large portion of blood volume in the portal bed. The late drop was attributed to the moribund state of the animal rather than the effect of portal vein ligation.

2. Ligation of the portal vein with a portacaval shunt produced no change in systemic blood pressure.

3. Incomplete occlusion of the portal vein caused no change in blood pressure but did produce changes that have been previously described [5] in the liver and in the collateral bed of the portal system. There was a transient fall in blood pressure. However, the pressure returned to normal within a few days.

10. Peck, M., and Grover, R.: Personal communication to the authors.

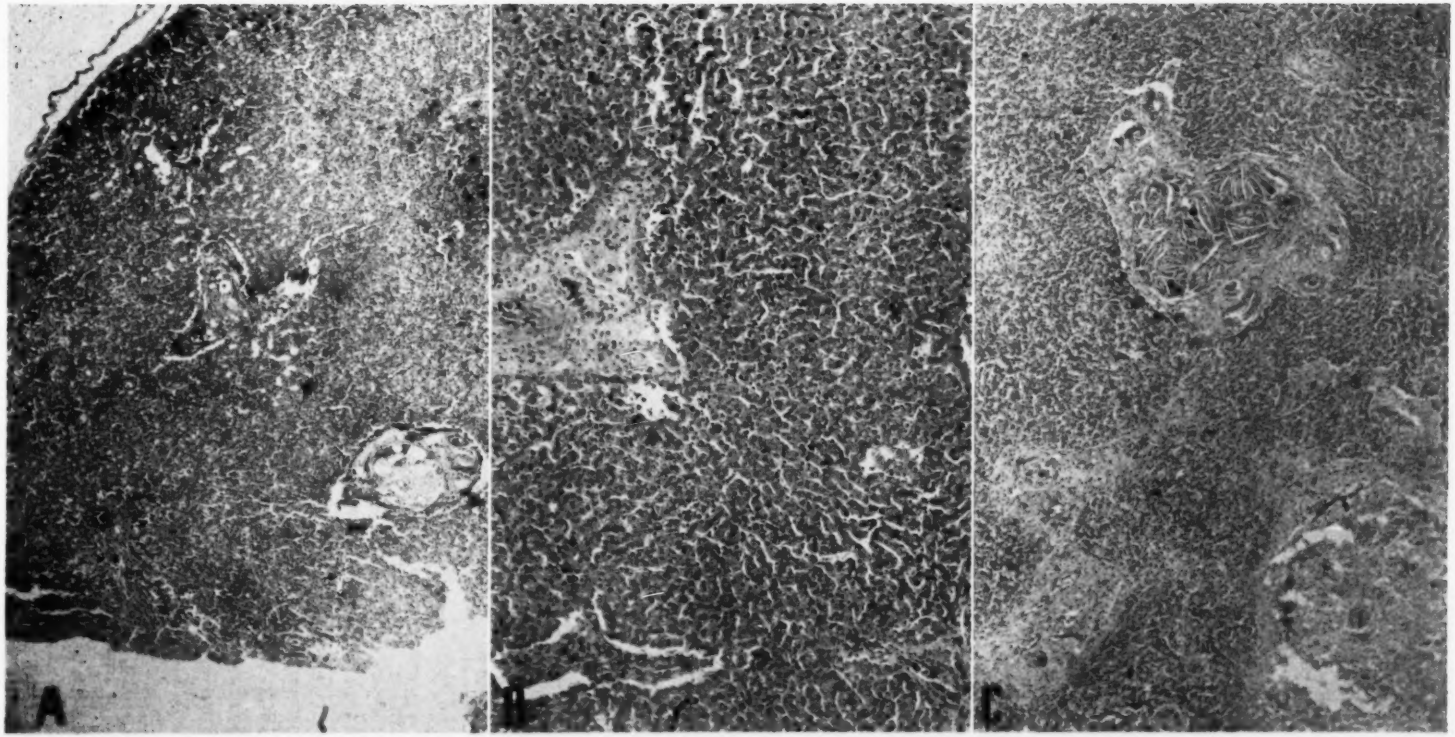

Fig. 3 (Dog B 89).—(*A*) Biopsy specimen of the liver one week after injection of 10 cc. of 0.9% dicetyl phosphate in olive oil into the portal vein, which had been constricted three weeks previously. Note early foreign-body reaction to oil. Hematoxylin and eosin stain; × 60. (*B*) Biopsy specimen taken four weeks later. Note early cirrhosis and abnormal liver lobules. Hematoxylin and eosin stain; × 100. (*C*) Biopsy specimen of the liver taken eight weeks after injection of 0.9% dicetyl phosphate in oil. Fairly well developed cirrhosis secondary to foreign body granuloma. Hematoxylin and eosin stain; × 60.

4. Incomplete occlusion of the portal vein and simultaneous ligation of the hepatic artery produced no change in systemic blood pressure.

5. Ligation of the hepatic artery first and then incomplete occlusion of the portal vein with a polyethylene band, three to six weeks later, produced no changes in systemic blood pressure.

III. Passive Hyperemic Cirrhosis and Hepatic Fibrosis.—1. Passive hyperemic cirrhosis produced by incomplete occlusion of the inferior vena cava with a strip of polyethylene film caused a temporary fall in blood pressure, then ascites with slight portal hypertension. Arterial pressure returned to normal within a few days, and portal pressure fell gradually. Ligation of the hepatic artery 8 to 10 weeks following this procedure caused the portal pressure to return to normal, with amelioration of the ascites. The blood pressure remained constant.

2. The injection of 0.9% dicetyl phosphate in olive oil into the portal vein caused death within 24 hours. Cirrhosis produced by the injection of this material in the partially occluded portal vein produced no change in systemic blood pressure, and the animals survived (Fig. 3).

3. The injection of dicetyl phosphate in olive oil into the hepatic artery produced a precipitous fall in blood pressure, with death in from 6 to 48 hours from hepatic insufficiency. If the hepatic artery was ligated and then, three to six weeks later, dicetyl phosphate injected, the animal survived. However, there was no change in systemic blood pressure.

Group B—Hypertensive Dogs

Dogs were made hypertensive by unilateral nephrectomy, envelopment of the opposite kidney with silk, according to the method of Page and co-workers,[11] and the subsequent application of a Goldblatt clamp on the renal artery. All dogs were observed until the pressure had become stabilized at a high level. However, the recording of blood pressure by the intra-arterial manometer is subject to great variation in hypertensive dogs. We have observed that there are deviations from day to day, the time of day, the relation to feeding, and the seasons. Moreover, there may be a gradual fall in blood pressure with the passage of time. The humoral mechanism of hypertension may be determined by a study of angiotonin units, as described by Shipley, Helmer, and Kohlstaedt,[3] although the actual relationship of the amount of angiotonin and the degree of hypertension is not proved.

I. Hepatic Artery Ligation.—1. Effects of Ligation of the Hepatic Artery in Hypertensive Dogs: (*a*) Ligation of the hepatic artery at the celiac axis caused an increase in blood pressure and a slight rise in angiotonin units in the majority of dogs. This would indicate that ligation of the hepatic artery close to its origin either does not effect the amount of substrate produced by the liver or actually increases it. We were particularly interested in this phase of the subject during the course of our cirrhosis experiments because we considered this finding as added evidence that ligation of the hepatic artery at its origin may improve the function of the liver, although the blood pressure was unaffected by the same procedure in normotensive dogs (Fig. 4).

11. Page, I. H.; McSwain, B.; Knapp, G. M., and Andrus, W. D.: The Origin of Renin-Activator, Am. J. Physiol. **135**:214-223 (Dec.) 1941.

(*b*) As a result of ligation of the hepatic artery close to the liver, all animals died within 24 to 48 hours of liver necrosis or gangrene of the gall bladder whether penicillin was used or not. If cholecystectomy was done, peritonitis did not follow. The blood pressure fell rapidly until death, owing to the moribund state of the animals, a phenomenon which occurs in pyrogenic and prelethal states from any cause.

(*c*) Ligation of the hepatic artery at its origin and then, in six weeks to four months, ligation of collaterals close to the liver caused death of 90% of all animals within five days after the collaterals were tied. The blood pressure falls as described under *b*. The angiotonin units remained the same (Fig. 5).

II. Portal Vein Ligation and Occlusion.—1. Ligations of the portal vein in one hypertensive dog caused death within 24 hours. There was a fall in blood pressure which we thought was due to the moribund state of the animal.

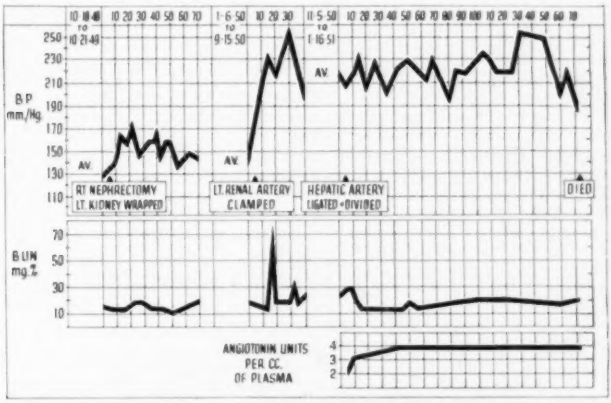

Fig. 4 (Dog 52 B 18, admitted Oct. 18, 1948).—Effects of hepatic artery ligation in hypertensive dog. Average blood pressure during preliminary observation period (Oct. 11, 1948, to Oct. 21, 1949) was 130 mm. Hg. Following right nephrectomy and constriction of left kidney with a silk envelope, there was a prompt rise in blood pressure. Pulse rate remained stable at about 80 beats a minute. Blood urea nitrogen ranged from 18 to 20 mg. per 100 cc., and weight stayed at about 13 kg. Animal was observed for about 11 months, then a Goldblatt clamp was applied to left renal artery. This resulted in an enormous increase in blood pressure up to 260 mm. Hg, but it soon fell to 215 mm. Hg. Pulse rate increased to 200 beats a minute. Blood urea nitrogen rose to 75 mg. per 100 cc. Body weight remained constant.

On Jan. 16, 1951, which was about four months after constriction of renal artery, hepatic artery was ligated and divided. Blood pressure rose gradually to 260 mm. Hg and remained high until shortly before death. Pulse rate remained at about 120, blood urea nitrogen at about 20 mg. per 100 cc., and weight at about 15 kg. Angiotonin units rose from 2.3 to 4 and remained elevated until dog died five and one-half months later. At autopsy, carcinoma of lung was found, with nodules in pleura, mediastinum, and pericardium.

2. Ligation of the portal vein with immediate portacaval shunt cause no change in systemic blood pressure or angiotonin units.

3. Incomplete occlusion of the portal vein with periportal dicetyl phosphate injection or with a polyethylene band causes a transient ascites and the formation of some collateral circulation. There were no esophageal varices and only a slight increase in portal pressure. There was a transient fall in systemic blood pressure but no significant fall in angiotonin units (Fig. 6).

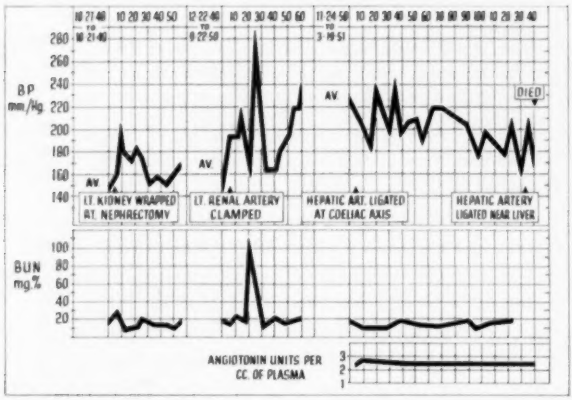

Fig. 5 (Dog 52 B 19, admitted Oct. 27, 1948).—Hypertensive dog. Effects of ligation of the hepatic artery at its origin and four months later ligation of collaterals close to the liver. Blood pressure average 150 mm. Hg during preliminary period of observation. Following right nephrectomy and envelopment of left kidney with silk, blood pressure promptly rose to 205 mm. Hg. Pulse rate increased from 80 beats a minute to 120. Blood urea nitrogen remained at about 20 mg. per 100 cc., and weight at about 11 kg. After 11 months, left renal artery was partially occluded with a Goldblatt clamp. Blood pressure rose to 288 mm. Hg; pulse rate increased to 118 and blood urea nitrogen to 110 mg. per 100 cc. There was a slight rise in weight to 15 kg., but this soon returned to 11 kg.

Six months later, hepatic artery was ligated at celiac axis. Blood pressure rose to 240 mm. Hg. Pulse varied between 100 and 120 beats a minute. Blood urea nitrogen remained at about 20 mg per 100 cc. Weight again increased to 15 kg. Angiotonin units remained constant at 2.6.

One hundred forty days later, branches of hepatic artery were tied near the liver. This resulted in a prompt fall of blood pressure, and animal died of hepatic necrosis.

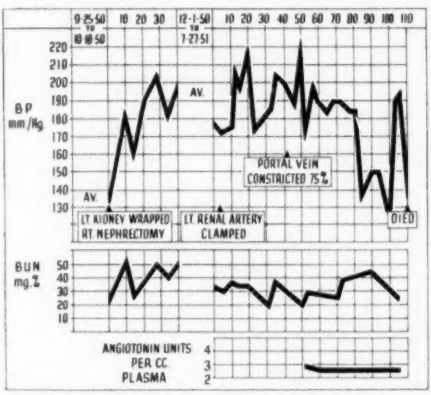

Fig. 6 (Dog P 579, admitted Oct. 25, 1950).—Hypertensive dog. Effect of constriction of portal vein with polyethylene band. Blood pressure averaged 130 mm. Hg during observation period, Sept. 25 to Oct. 18, 1950. Blood urea nitrogen was 20 mg. per 100 cc.; pulse rate, 80 beats a minute; weight, 12 kg. After right nephrectomy and application of a silk envelope around left kidney, blood pressure rose to 200 mg. Hg, blood urea nitrogen to 55 mg. per 100 cc., and pulse rate to 120 beats a minute. Weight remained constant.

Blood pressure fell slowly during the seven-month observation period (Dec. 1, 1950, to July 27, 1951) to 178 mm. Hg. At this time, left renal artery was constricted with a Goldblatt clamp. Blood pressure rose to 225 mm. Hg; pulse rate increased from 90 beats a minute to 120 beats a minute; blood urea nitrogen varied between 20 and 30 mg. per 100 cc.

Forty days later, portal vein was constricted about 75% with a polyethylene band. This resulted in a decline in blood pressure to 140 mm. Hg for about 20 days, then a rise to 190 mm. Hg. Blood urea nitrogen rose to 45 mg. per 100 cc., pulse rate decreased to 80 beats a minute, and angiotonin units declined from 3 to 2.5 units. Animal died of a small perinephric abscess and pyelonephritis.

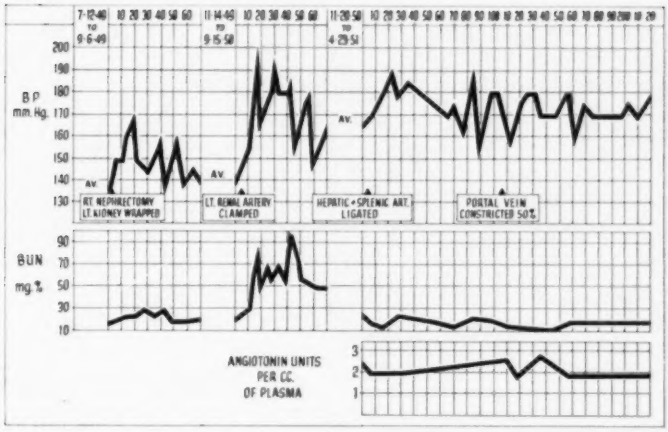

Fig. 7 (Dog 52 B 10, admitted July 12, 1949).—Hypertensive dog. Effects of ligation of the hepatic and splenic arteries and then four months later constriction of portal vein. Average blood pressure was 130 mm. Hg during two-month preliminary observation period. After right nephrectomy and the envelopment of left kidney with a silk envelope, blood pressure rose to 170 mm. Hg. Pulse rate varied between 80 and 100 beats a minute. Blood urea nitrogen varied between 20 and 30 mg. per 100 cc. Weight remained at 10 kg.

Two months later, blood pressure had returned to 140 mm. Hg. At this time, left renal artery was constricted. Blood pressure promptly rose to 200 mm. Hg; pulse rate increased to 120, and blood urea nitrogen rose to 100 mg. per 100 cc. Weight increased to 12 kg. During the ensuing five months, blood pressure averaged 165 mm. Hg.

After ligation of hepatic and splenic arteries, blood pressure rose to 190 mm. Hg and remained relatively constant. Pulse rate decreased to 90 beats a minute, blood urea nitrogen stayed at about 20 mg. per 100 cc., and weight 12 kg. There was a gradual rise in angiotonin units from 2 to 2.5.

One hundred ten days later, portal vein was constricted to about 50% of its lumen. This did not change level of blood pressure, but pulse rate increased to 110 beats a minute. Blood urea nitrogen remained at 20 mg. per 100 cc., and there was no change in animal's weight. Angiotonin units fell to 1.8.

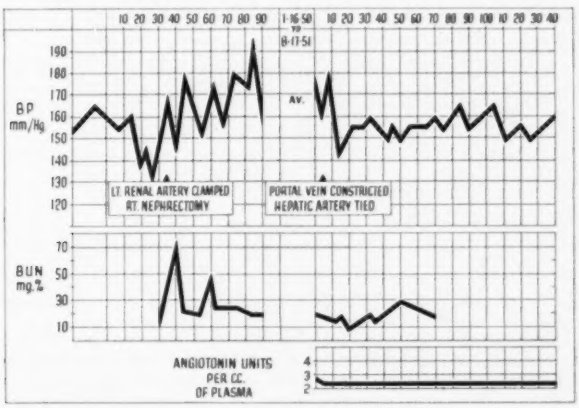

Fig. 8 (Dog 52 B 21, admitted Oct. 19, 1949).—Effects of portal vein constriction with simultaneous ligation of hepatic artery. Note variations in blood pressure prior to constiction of left renal artery, also daily variations thereafter. There is a prompt rise in blood pressure following application of Goldblatt clamp, accompanied by a rise in pulse rate from 80 to 120 beats a minute, and an increase in blood urea nitrogen. Pulse rate returned to normal in about 15 days and remained so throughout period of observation. Same was true of blood urea nitrogen. Weight remained constant at 12 kg. Angiotonin units averaged 2.3 per cubic centimeter of plasma.

After a period of observation of 20 months, portal vein was constricted with a polyethylene band and hepatic artery ligated at the same time. Note prompt fall in blood pressure from 180 to 140 mm. Hg and then a rise to 160 mm. Hg. Blood urea nitrogen rose from 10 to 30 mg. per 100 cc. and then returned to normal of 20 mg. per 100 cc. Angiotonin units decreased slightly. Pulse rate and weight remained constant.

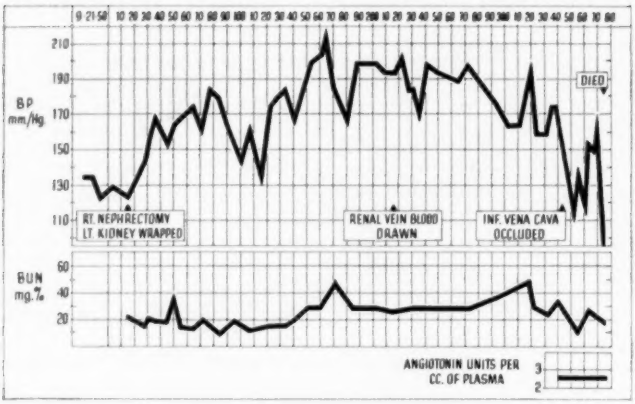

Fig. 9 (Dog PB 77, admitted Sept. 21, 1950).—Hypertensive dog. Effects of occlusion of inferior vena cava with polyethylene band above diaphragm. Average blood pressure was 125 mm. Hg during period of preliminary observation. Following right nephrectomy and envelopment of left kidney with silk, blood pressure rose gradually to 230 mm. Hg. Pulse rate varied between 80 and 100 beats a minute. Blood urea nitrogen rose to 50 mg. per 100 cc. but slowly decreased to 30 mg. per 100 cc. Weight was 15 kg.

Eleven months later, inferior vena cava was constricted with a polyethylene band to about 50% of its diameter. Within 10 days, blood pressure decreased to 110 mm. Hg, and pulse rate increased to 140 beats a minute. Blood urea nitrogen rose to 70 mg. per 100 cc. There was a 3 kg. loss in weight despite accumulation of ascitic fluid. Angiotonin units remained constant at 2.4. Animal died 20 days after this procedure. Autopsy disclosed pneumonia with empyema and pericarditis.

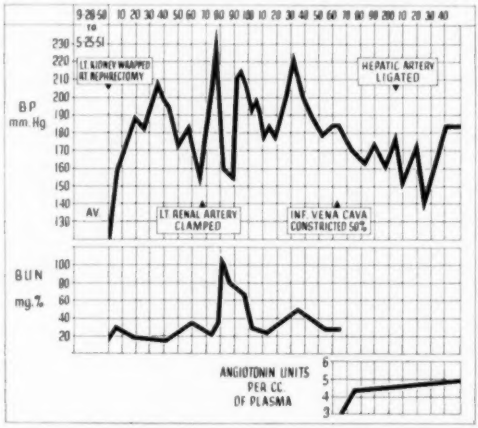

Fig. 10 (Dog P 582, admitted Oct. 28, 1950).—Hypertensive dog. Incomplete occlusion of inferior vena cava with polyethylene band and then 40 days later ligation of hepatic artery. This dog was observed from Sept. 28, 1950, to May 25, 1951, and averaged blood pressure was recorded as 127 mm. Hg. Following right nephrectomy and envelopment of left kidney with silk, blood pressure rose to 210 mm. Hg. Pulse rate increased from 80 to 140 beats a minute. Blood urea nitrogen remained at 20 mg. per 100 cc. Weight stayed at 10 kg.

Seventy days later, blood pressure had decreased to 160 mm. Hg, and left renal artery was constricted. Note great rise in blood pressure to 237 mm. Hg and concomitant rise in blood urea nitrogen to 120 mg. per 100 cc. Pulse rate increased to 120. Weight remained at 10 kg.

Three months later, inferior vena cava was constricted to about 50% of its diameter with a polyethylene film. Note gradual decline in blood pressure to 165 mm. Hg. Pulse rate and blood urea nitrogen returned to normal levels and remained constant. Weight did not vary.

Forty days after occlusion of vena cava there was a great amount of ascites, and many venous collaterals had formed on abdominal wall. At this time, hepatic artery was tied. Note gradual rise in blood pressure to 185 mm. Hg. Pulse rate decreased to 100 beats a minute, and blood urea nitrogen remained at 20 mg. per 100 cc. There was no change in weight. Angiotonin units rose from 3 to 5 per cubic centimeter of plasma.

4. Ligation of the hepatic artery first and then constriction of the portal vein by a cellophane® band, three to six months later, produced either no effect in systemic blood pressure or a slight rise. The angiotonin units were not significantly altered (Fig. 7).

5. Ligation of the hepatic artery at its source and constriction of the portal vein at the same time produced a fall in blood pressure followed by a slight but persistent rise. The angiotonin units were not significantly altered (Fig. 8).

III. Passive Hyperemic Cirrhosis and Hepatic Fibrosis.—1. Passive hyperemic cirrhosis produced by incomplete occlusion of the vena cava with a polyethylene band, as previously described, produces a fall in systemic pressure. The angiotonin units remain constant (Fig. 9).

2. Incomplete occlusion of the vena cava, as described in the preceding paragraph, then ligation of the hepatic artery is followed two to three weeks later by a rise in systemic pressure and in angiotonin units (Fig. 10). This result was not obtained in normotensive cirrhotic dogs.

3. The injection of dicetyl phosphate into the portal vein, as previously described, does not produce a change in systemic blood pressure or in angiotonin units.

4. The injection of dicetyl phosphate in the hepatic artery after preliminary ligation does not produce a change in systemic pressure or in angiotonin units.

5. Anesthesia induced by thiopental (pentothal®) sodium injected intraperitoneally without surgery caused no permanent changes in the systemic blood pressure or in the angiotonin units.

SUMMARY OF EXPERIMENTAL FINDINGS

Our experiments on normotensive, hypertensive, and cirrhotic dogs show that there is no long-term relationship between portal hypertension and systemic blood pressure per se. The only group of animals in which we were able to produce a sustained fall in systemic blood pressure were those that had a passive hyperemic cirrhosis of the liver induced by incomplete ligation of the vena cava with a polyethylene band. These animals failed to show a decrease in angiotonin units. The rise in systemic blood pressure two to three weeks after ligation of the hepatic artery close to the celiac axis in cirrhotic hypertensive animals led us to believe that perhaps this procedure restored some of the function of the liver and thereby facilitated the production of renin substrate. However, since hepatic arterial ligation in ascitic and hypertensive dogs caused a diminution of ascites, the rise in arterial pressure may have resulted from an interruption of the continued plasmapheresis or a change in cation (sodium and potassium) relationships. Moreover, vena cava occlusion increases renal vascular stasis, theoretically increasing the production of renin. However, there was no increase in blood pressure in normotensive cirrhotic dogs after hepatic arterial ligation.

CIRRHOSIS OF THE LIVER AND SYSTEMIC HYPERTENSION IN MAN

It has long been known that the combination of Laennec's cirrhosis and systemic hypertension is unusual. Statistics vary, but in our own clinic the incidence of cirrhosis in hypertension is less than 3%, and the incidence of hypertension in cirrhosis is less than 2%. We have observed two patients with cirrhosis and hyper-

tension. Both were treated by ligations of the hepatic and splenic arteries before we knew the dangers of this procedure in hypertensives. The first patient has been previously reported on.[12]

M. N., a white woman aged 58, was admitted to the Indianapolis General Hospital on June 22, 1951, complaining of abdominal swelling and edema of the ankles. She had been receiving medical treatment for the past three years, but her abdomen had increased so much in size within the previous three months that it was difficult for her to breathe. Her systolic blood pressure was 230 and the diastolic was 110. The heart was greatly enlarged, and a loud systolic murmur could be heard over the apex. X-ray examination showed an enlarged heart, but all other studies gave normal results. There were no varices at the time of the barium swallow, although the patient stated that on one occasion she had vomited a slight amount of blood. The laboratory findings were as follows: total proteins 6.5 gm. per 100 cc., 3.6 gm. of albumin, and 2.9 gm. of globulin. Results of the sulfobromophthalein sodium showed 1.5% retention at 30 minutes and 0.3% retention at 45 minutes; the prothrombin time, 100% of normal, and the nonprotein nitrogen, 49 mg. per 100 cc. A biopsy of the liver showed portal cirrhosis. We thought that this was an early case which should yield excellent results from surgery. On June 29, 1951, the abdomen was explored through a transverse incision. The portal pressure was 345 mm. of citrate. After the hepatic and splenic arteries were ligated close to the celiac axis, the pressure fell to 202 mm. of citrate. On the second postoperative day the patient became stuporous, and the blood pressure fell to 100 systolic and 60 diastolic. She died of "hepatic crisis" in spite of measures we had used successfully in similar cases. At autopsy the ligatures were checked and found to be close to the celiac axis on the common hepatic artery and on the splenic artery. There were small areas of hemorrhage in the capsule of the spleen and of the liver and around the esophagus and in the pericardium. There was advanced portal cirrhosis, atelectasis of both lower lobes of the lungs, and advanced coronary atherosclerosis.

The second patient, V. P., was a man 65 years of age, who came to the Indianapolis General Hospital complaining of ascites. He stated that 1 to 2 gal. of fluid had been removed from his abdomen at bimonthly intervals during the past two years. The principal physical findings were a loud systolic murmur, heard best at the base of the heart and transmitted to the left, and ascites. His blood pressure in the right arm was 228/100 and in the left 236/115. Laboratory studies gave the following results: The blood contained 3,820,000 erythrocytes, with 11.4 gm. of hemoglobin, and 8,100 leucocytes, with 78% neutrophiles and 22% lymphocytes. The prothrombin time was 80% of normal; blood urea nitrogen measured 19 mg. per 100 cc. The sulfobromophthalein sodium test showed 28.1% retention at the end of 30 minutes and 19.3% retention at the end of 60 minutes. Reactions to the Kahn and Kline tests were negative. The total protein was 6 gm. (3.19 gm. of albumin and 2.81 gm. of globulin); the volume of packed red blood cells was 39 per 100 cc. Urinalysis showed a specific gravity of 1.014, a 2+ reaction for albumin, and a negative reaction for sugar. The sodium excretion in the urine was 18.4 mEq. in 24 hours, and a repeat examination showed 26.9 mEq. in 24 hours; potassium was 18.9 mEq., and a second test showed 27.6 mEq. per 24 hours. The blood sodium was 137 mEq., potassium was 4.36 mEq., and chlorides were 108 mEq. The circulation time (ether) arm to lung was approximately 15 seconds and (magnesium sulfate) arm to tongue approximately 26 seconds. At the time of surgery, Sept. 25, 1951, the portal pressure was 398 mm. of citrate. A 5% solution of fluorescein sodium was injected into the hepatic artery and was seen in the hepatoduodenal ligament in 10 seconds. The hepatic artery was tied close to the celiac axis, as were also the splenic and left gastric arteries. Portal pressure after the ligation was 294 mm. of citrate. A biopsy of the liver showed portal cirrhosis. The immediate postoperative course was uneventful. On the fifth postoperative day the patient complained of weakness; his skin was clammy; the blood pressure was 150/100, and the pulse rate, 180 to 200 and irregular. On the sixth day he became comatose; the blood pressure was 95/60 and, as in the preceding patient, there was also anuria and death from "hepatic crisis."

12. Berman, J. K., and Hull, J. E.: Hepatic, Splenic, and Left Gastric Arterial Ligations in Advanced Portal Cirrhosis, A. M. A. Arch. Surg. **65**:37-65 (July) 1952.

Tests showed liver function normal in Patient M. N. and not greatly altered in Patient V. P. This would lend credence to the prevalent hypothesis that such a liver can produce sufficient substrate to combine with renin and elevate the blood pressure to the level which these patients had. In cirrhotic patients with hypertension we must conclude that sufficient oxygen reaches the ischemic liver cells to enable them to produce enough renin substrate to maintain the elevated blood pressure. This is possible because of the ratio of pressures between the hepatic arterial and portal venous components. Normally the hepatic arterial pressure is 120 mm. of mercury, and the portal venous pressure is 8 to 12 mm. of mercury, a ratio of about 10 to 1. In most cirrhotic patients portal pressure is doubled, but arterial pressure remains the same. Therefore, the ratio becomes greatly reduced, amounting to about 5 to 1. Livers with such moieties of circulation are better able to withstand the sudden decrease of arterial pressure after ligation, although there is a temporary insufficiency of arterial blood as manifested clinically by what we have termed "hepatic crisis." In patients with hypertension the ratio of pressures is again about 10 to 1 but on a much higher level. In Patient M. N. the portal pressure was 26 mm. of mercury, and the systolic blood pressure was 230 mm. of mercury. In Patient V. P. the portal pressure was 31 mm. of mercury and the systemic pressure 236 mm. of mercury. The cirrhotic liver cells under such conditions obtain their oxygen through excessive arterial pressure, which they demand to remain viable. They apparently cannot withstand the sudden drop to near zero pressure after hepatic arterial ligation, and therefore the "hepatic crisis" becomes insurmountable. Conceivably, if the liver with such moieties could survive long enough for collaterals to form, it would benefit more from hepatic arterial ligation than would the cirrhotic liver in normotensive patients. The sequence may be compared to the anuria which follows prolonged hypertension in patients with ischemic kidneys and high blood pressure. Indeed, the anuria in hepatorenal syndrome following hepatic-splenic arterial ligation may be due to the fall in blood pressure which usually occurs in this crisis. Liver cells seem to remain viable long enough for collaterals to form if they become acclimated to the ratio of pressures which usually occur in normotensive cirrhotic persons. Perhaps if we could lower the systemic blood pressure gradually so that vascular compensation would occur prior to surgery, the operation could be successfully employed. The patients' deaths were due to hepatic and renal insufficiency in both instances. It would seem from careful observation of these two patients that the coincidence of systemic hypertension with cirrhosis of the liver implies a fairly large amount of normal parenchyma; otherwise, renin substrate could not be produced in sufficient amount to cause the systemic hypertension. However, we realize that two cases are not statistically significant.

COMMENT

Our experimental studies indicate that any great deterioration and diminution of liver function may interfere with the production of renin substrate. Hepatic function is not impaired in our experimental animals with passive hyperemic cirrhosis to the degree which is observed in portal cirrhosis in man.

Portal cirrhosis associated with a great amount of liver enfeeblement, as determined by laboratory studies, implies decreased liver function, although many badly damaged livers may yield normal laboratory results. Such livers are unable to produce a sufficient quantity of renin substrate to cause hypertension. On the other

hand, when hypertension complicates the portal cirrhosis, reactions to liver tests are very close to normal. The ascites in these patients is a manifestation of hepatic vascular and lymphatic decompensation without great functional deterioration. It is possible that the continuous plasmapheresis in ascites may prevent an increase in arterial pressure due to diminished blood volume. However, there has been no elevation in blood pressure following hepatic-splenic arterial ligation with amelioration of ascites in the 22 normotensive cirrhotic patients on whom we have operated.

The degree of organic change in the arterioles and kidneys may be a factor in patients with advanced portal cirrhosis. If the hypertension preceded the cirrhosis and if it remained, then organic changes could occur in the arterioles and kidneys which might be irreversible. Hypertension would persist whether the liver was capable of producing renin substrate or not. However, the onset of cirrhosis is so insidious that it would be difficult to acertain its time relationship to the hypertension. Moreover, the relatively small change in liver function makes the detection of priority even more difficult. In our patients, organic changes characteristic of late hypertension were not demonstrable. The arterial hypertension is apparently necessary to keep the liver parenchyma functioning; therefore a vicious circle is established in which hypertension is apparently necessary to keep the liver cells viable, yet they in turn are able to produce a sufficient amount of substrate to cause a persistence of the hypertension.

CONCLUSIONS

1. The experiments we have described lend credence to the theory that the liver does play a role in the cause and perpetuation of certain types of systemic hypertension. However, the counterpart of that observed in man is difficult to produce in the experimental animal.

2. Experimental passive hyperemic cirrhosis with vascular decompensation may be improved by ligation of the hepatic and splenic arteries close to the celiac axis. The increase in blood pressure after two to three weeks in hypertensive animals may imply improved hepatic funtion after these ligations, although the amount of angiotonin is not greatly elevated.

3. Portal cirrhosis in man, complicated by systemic hypertension, should not be treated by ligation of the hepatic and splenic arteries because the hepatic cells are dependent on the hypertension to sustain their viability, and, after the hepatic artery is ligated in hypertensives, there is a fall in blood pressure during the "hepatic crisis." After this period the systemic blood pressure may rise to levels higher than those observed before operation.

DISCUSSION

ON PAPERS OF RACK, MINCKS, AND SIMEONE; SHUMACKER AND KING, AND BERMAN AND HULL

DR. WILLIAM D. HOLDEN, Cleveland: Dr. Rack's paper concerning the occurrence of esophageal varices in the absence of portal hypertension is very noteworthy. I think all of us when we think of esophageal varices are very prone to associate them immediately with portal hypertension.

Since Dr. Rack first presented me with the data he had, I have had an opportunity to look over the literature, and a remarkable number of patients have esophageal varices demonstrated roentgenographically and also demonstrated at autopsy. I think it is important to emphasize the

fact that varices do occur in the absence of portal hypertension, and we should note it and make a search for some other etiologic agent when portal hypertension as a result of cirrhosis is not apparent. I think it is also important to emphasize the fact that the majority of patients who have esophageal varices have them as a result of portal hypertension.

Varices apparently are both functional and anatomical in origin. It is very difficult to understand exactly how they develop in the absence of an elevated pressure within the portal system. We have observations, however, to the effect that these varices can be enormous and yet within a short time can disappear completely.

The association with coronary heart disease is very difficult to understand. It is true that in the absence of portal hypertension varices occur principally in older persons, perhaps as the result of the degenerative processes that occur in the connective tissues supporting the veins in the lower esophagus. When they do occur in such circumstances they usually are not limited to the lower third of the esophagus, and I think that is very helpful in differentiating the varices resulting from portal hypertension from those in which the origin is extremely obscure.

As far as the second paper is concerned, I should like to record one other factor in the pathogenesis of portal hypertension. We had the opportunity to manage an extremely unusual case just a short time ago, and probably we shall never see another like it.

The patient was a young girl with hypersplenism and obvious manifestations of portal hypertension. We missed the preoperative diagnosis, but at the operating table it was seen that she had a congenital arteriovenous fistula of the splenic vessels. It was removed satisfactorily, and we hope the patient is cured. It was so unusual that I thought it was worth bringing to your attention.

With regard to portal hypertension and the various shunt operations, I hesitate and am very reluctant to detract at all from the experimental procedures that surgeons are reporting throughout the country, and yet our experience with portal hypertension and the shunting procedures has not been as satisfactory as the experiences of many other surgeons in this country.

On numerous occasions, I am rather convinced in my own mind, from having taken pressures in various veins before and after creating a shunt, that during the course of the operation I have interrupted more collateral venous channels than I have actually created by doing the anastomosis. We don't have any long-term survivals, but there are many things about portal hypertension and the shunting procedures that are unsatisfactory.

The clinical evaluation of the patient, as pointed out by Dr. King, must be done over a long time if we are to evaluate these patients successfully. Years ago when splenectomy was being done for these patients, many of them got along well for a good many years, but at present I think we are all convinced that splenectomy is neither a desirable nor an effective operation.

As far as hepatic artery ligation for portal hypertension is concerned, we have had very little experience with it. However, in one patient with hepatic cirrhosis and a pressure of 450 mm. of water, we introduced a catheter into the portal vein and had a very adequate continuous recording of pressure. We clamped off the hepatic artery with a soft clamp. Repeatedly, we were unable to demonstrate any significant change in portal pressure. That observation is of only one patient. I don't know whether it is valid or not, but it would be very worth while to hear other comments about it.

Dr. William Altemeier, Cincinnati: I should like to emphasize a point briefly alluded to by Dr. Holden, the possible relationship of an arterial venous shunt between the hepatic, splenic, or left gastric arteries and the portal system in portal hypertension.

We have done 11 ligations of the hepatic and splenic arteries, or the hepatic, splenic, and left gastric arteries, in patients with portal hypertension and esophageal varices. In at least 4 of these 11, numerous small shunts appeared to pass between the hepatic and splenic arteries and the adjacent portal or splenic veins.

In addition, the hepatic and splenic arteries showed marked dilatation, thinning of the walls, and a readily palpable thrill, such as are found in other arterial venous aneurysms. The thrill could also be felt easily in the adjacent veins, including pools of large venous channels as far away as 6 in. (15.2 cm.) from the arteries. Also, the blood found in the portal vein is usually bright red.

In a fifth case in which a splenectomy was being done in a patient with esophageal varices, the splenic vein was inadvertently torn. Although the vein was quickly clamped on either side

of the laceration, it was temporarily impossible to control the bleeding completely. Numerous small ostia of vessels were seen entering the side of the portal vein, and through these bright-red blood spurted.

Recently, we have sterilized a stethoscope and used it at the operating table to listen directly to the biphasic murmur.

I should also like to record another experience which we had with one of our patients who died about eight weeks after the hepatic and splenic arteries were ligated. A septic necrosis of the superior pole of the spleen developed, presumably due to ligation of the splenic artery. The large subphrenic abscess which resulted was the cause of the patient's death.

DR. GEORGE CRILE JR., Cleveland: I am in full agreement with Dr. King's remarks regarding the lack of value of the devascularization procedure and certainly also with the remarks by Dr. Holden regarding the incidence of esophageal varices in the complete absence of demonstrable portal hypertension. It is in such cases that the operation which I reported here two years ago is of value.

That operation, as some of you may remember, is a direct attack on the varices themselves, in which the esophagus is opened and the varices obliterated by running a nonabsorbable surgical (gut) suture throughout the lower third of the esophagus. This is technically an easy operation to do and so far has carried no mortality.

The eight cases that we reported two years ago have been followed two years longer. All these patients were frequent bleeders. The minimum number of transfusions which they had was about 20, and some of them had as many as 300 transfusions. Most of them were bleeding at intervals of anywhere from a few weeks to every few months.

These patients were all young people; in none of them was the condition due to liver disease, and many of them had no demonstrable portal hypertension at the time of operation. Most of them had had their spleens removed. These patients have done extremely well. One patient, who was reported before as having bled again after operation, in the presence of a demonstrable gastric ulcer has continued to have ulcer pain and to bleed periodically but not severely. We do not know whether the bleeding is from the varices or from the ulcer.

Another patient had over 300 transfusions and has had only one small hemorrhage since operation. The entire esophagus in that case was involved. Our operation involved the lower third. The other patients have had no bleeding since.

I believe that in those patients who are not amenable to the operation of portacaval shunts or lienorenal anastomoses the obliteration of the varices is a much simpler and safer procedure than the resection of the esophagus and stomach, which carries a high mortality and a rather high morbidity.

DR. JOHN H. GRINDLAY, Rochester, Minn.: I think most of us would agree that there is more confusion than erudition about the liver and the role of its blood supply. Studies such as Dr. Berman has reported have been done by my colleagues and me, but we aren't certain how they should be interpreted.

I thought you might be interested in some preliminary results of experiments we are doing on the effect of ligation of the hepatic artery in normal and in cirrhotic dogs. We have worked out a surgical technique which permits us to obtain hepatic venous blood from dogs at will, for periods as long as three or four weeks. The catheter is placed in a hepatic vein, but I shall not go into the technique here. At the same time, we place another catheter in the portal vein and another in the inferior cava.

We have learned that ligation of the hepatic artery does not seem to affect the oxygen saturation, the rate of blood flow leaving the liver, or the pressure in the hepatic veins. This seems to indicate that ligation of the hepatic artery does not materially change the rate of inflow of arterial blood.

When the hepatic branches are ligated close to the liver, on the other hand, there is a marked fall in the oxygen of hepatic vein blood. You will recall that dogs in which this is done die promptly unless given penicillin. Even if penicillin is administered, many of them die within three weeks of liver necrosis. However, if they get an arterial supply through their collateral system and recover, it appears that hepatic venous blood is about the same as in a normal dog. The pressure studies have revealed also that there is a strange type of pressure wave in the hepatic veins which is related to intrathoracic pressure.

I should like to comment on Dr. Rack's interpretation of the origin of varices. In dogs with portal obstruction we have been unable to find true submucosal varices in the esophagus. However, I have been able to produce such dilatations by obstructing the superior vena cava and excising the azygos vein. This indicates that in the dog, an animal whose normal position is horizontal, an impediment of flow through the portal collateral (by raising venous pressure at the point at which it must empty, namely, into the superior cava or azygos system) may produce varices.

Dr. Ormand C. Julian, Chicago: The results of the work in children by Drs. Shumacker and King are very much in keeping with ours in adults. We agree that the results of expectant treatment of esophageal hemorrhage associated with varicose veins and of splenectomy alone are poor. The devascularization operation offers nothing to these patients.

Gastroesophagectomy and shunt operations, in our hands, have remained as the tested procedures of choice.

It seems to us that, as pertaining to children, splenorenal anastomosis ought to be of principal value. We have taken the view, somewhat along the line of Dr. Rack and Dr. Simeone, that esophageal varices do not necessarily reflect a continuous portal hypertension. We feel that the portal hypertension, which is demonstrated by the presence of the varices, may disappear in certain circumstances in early cirrhosis when edema and fat infiltration of the liver produce a temporary obstruction of the flow of blood through the liver. In children the essential cause of portal hypertension and esophageal varices is extrahepatic, the pressure will remain up, and the shunts open. In cases in which the hypertension is fleeting, the flow through the shunt may be reduced to such an extent during the period of diminished pressure that the shunt will become permanently nonfunctioning. This may account for a few of the poor results that have been obtained in our series and others in which splenorenal anastomosis was used in cirrhotic patients.

We have three children in our series with portal hypertension in the age group reported by Drs. King and Shumacker. Each one of these has been subjected to a splenorenal anastomosis, and in follow-ups of 14, 20, and 24 months in these three patients there has been no recurrence of bleeding. These patients showed normal liver functions and are thought to have extrahepatic portal blocks.

Dr. Philip F. Partington, Cleveland: A patient who was cirrhotic, with postnecrotic cirrhosis, on operation had a portal pressure of only 10 cm. of water. Before operation this patient was bleeding continuously and after operation continued to bleed until death, one month later.

[Slide] This is the usual mechanism for the production of esophageal varices. You can see the obstructed portal vein, obstructed at the portal fissure, and the coronary vein going up and communicating with the esophageal varices. This film was made after postoperative injection of a small polyethylene catheter which was left in an omental vein for regional heparization following portacaval anastomosis.

We have done several of these studies, at operation as well. It is an easy procedure, and I suggest that such studies at the time of operation might shed further light on this difficult problem.

RISK OF URGENT SURGERY IN PRESENCE OF MYOCARDIAL INFARCTION AND ANGINA PECTORIS

F. A. dePEYSTER, M.D.
O. PAUL, M.D.
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GENERALLY it is conceded that a greater operative risk attends the patient with coronary disease than is faced by the person with a normal heart. The mortality rate in surgical patients with myocardial infarction has been variously reported [1] from 5.2 to 44.5%. The unrelenting progress of medical science reflected in the increased life span necessarily brings to the surgeon a greater number of elderly patients with degenerative cardiac changes. Among these patients are those with evidence of severe angina pectoris or myocardial infarction harboring coexistent disease requiring urgent surgery. Usually these patients have some painful or malignant process in which delays in surgical therapeusis are costly, inviting reduced survival rates or even denying significant prolongation of a pain-free existence. In essence, the earliest auspicious application of a definitive operation designed to obviate these undesirable elements is implied by the term "urgent surgery." In most instances the operation is of major magnitude in which the pleural or peritoneal cavity is entered.

The medical literature does not abound in references relative to the operative risk in the presence of severe angina or myocardial infarction, and further study is needed in this direction. Indeed, it has been stated that progress in determining operability has lagged behind progress in general surgery.[2] The difficulty is obvious in obtaining significant statistical data concerning patients with these degenerative changes subjected to surgery. Only by the combined experience of various groups can one approach the problem with some degree of statistical validity. The utilization of uniform standards and criteria in studying these patients would appear to be essential.

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

From the Departments of Surgery and Medicine of the Presbyterian Hospital, and the Department of Surgery of the Research and Educational Hospitals, University of Illinois.

1. Hannigan, C. A.; Wrobiewski, F.; Lewis, W. H. Jr., and LaDue, J. S.: Major Surgery in Patients with Healed Myocardial Infarction, Am. J. M. Sc. **222**:628, 1951. Morrison, D. R.: The Risk of Surgery in Heart Disease, Surgery **23**:561, 1948. Butler, S.; Feeney, N., and Levine, S. A.: The Patient with Heart Disease as a Surgical Risk: Review of 414 Cases, J. A. M. A. **95**:85, 1930.

2. Cole, W. H.: The Need for Greater Accuracy in the Determination of Operability, Surg., Gynec. & Obst. **94**:248, 1952.

PURPOSE OF STUDY

Our objectives are twofold: (1) to evaluate factors that have influenced the mortality rate of patients with severe angina pectoris or myocardial infarction who have been subjected to urgent surgery, and (2) to seek means which may reduce the operative risk in future surgical patients with similar cardiac complications.

MATERIAL AND METHODS

The material reviewed consists of the records of 33 patients with myocardial infarction and 7 patients with severe angina pectoris, all of whom were subjected to urgent surgery. The cardiac pathology described in the records existed prior to or occurred during operation, or was experienced by the patient in the convalescent period. The term "convalescent period" includes the immediate 31 postoperative days, or the period of hospitalization, whichever was longer.

Diagnostic Criteria for Myocardial Infarction.—Diagnostic criteria for anterior, posterior, and septal infarctions follow.

I. Anterior Myocardial Infarction

(*a*) Old

1. Q wave 0.04 second or more in duration in any lead from VI through V_6
2. Q-S complex in any precordial lead to the left of the transition zone if accompanied deep (greater than 2.0 mm.) symmetrical T wave inversion

(*b*) Recent

1. Same as I *a*
2. History of chest pain with serial changes in the left precordial leads without significant Q waves but with change of one or more T waves from upright to inversion of at least 2 mm.
3. Similar history and changes in the precordial leads to the right of the transitional zone provided the T waves showed an inversion of 4 mm. or more.

II. Posterior Myocardial Infarction

(*a*) Old

1. Q or Q-S in lead aV_F 0.04 seconds or more in duration except with extreme horizontal hearts
2. If no aV_F available, Q or Q-S in lead III 0.04 seconds or more in duration with symmetrical inversion of T_3 2 mm. or more in depth associated with inversion of T_2

(*b*) Recent

1. Same as II *a*
2. History of chest pain with serial changes in lead aV_F without significant Q or Q-S complexes but with change of T from upright to inverted, with or without S-T segment elevation, lasting over 48 hours, associated with appearance of fever, and/or elevation of sedimentation rate, and/or elevation of white blood cell count
3. If no aV_F available, similar history with serial changes in leads II and III without significant Q or Q-S complexes but with change of T_3 from upright to symmetrical inversion 2 mm. or more in depth, associated with flattening or inversion of T_2, lasting over 48 hours, and coinciding with appearance of fever, and/or elevation of sedimentation rate, and/or elevation of white blood cell count

III. Septal Infarction

(*a*) May be combined with picture of anterior or posterior infarction given above

(*b*) History of chest pain associated with appearance of complete bundle branch block, lasting over 48 hours, coinciding with appearance of fever, and/or elevation of sedimentation rate, and/or elevation of white blood cell count

In the absence of changes in the electrocardiogram, cases were included as myocardial infarction only if sizable infarcts were proved to be present at autopsy. It is recognized that no criteria are absolutely infallible in the diagnosis of myocardial infarction. The above standards are considered to provide reasonable guides to the exclusion of minor changes of dubious importance.

Diagnostic Criteria for Angina Pectoris.—A diagnosis of angina pectoris was accepted if the patient gave a history of repeated attacks of substernal chest pain, with or without shoulder or arm radiation, coming with effort and emotion and promptly relieved by rest and administration of glyceryl trinitrate.

Ninety-six patients bearing the diagnosis of coronary heart disease associated with surgical procedures were selected. Of these 56 were discarded either because of an equivocal history of angina or because the laboratory evidence of myocardial infarction failed to meet the criteria outlined above. Most patients fell into the latter group. In some instances the original electrocardiographic tracings or unequivocal evidence of specific cardiac disease could not be

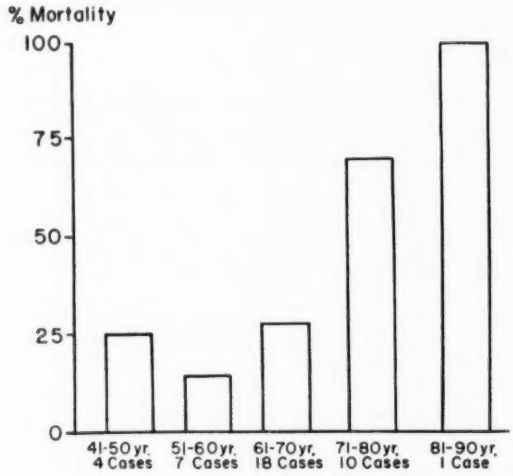

Chart 1.—Age and mortality rate. Relationship of age to mortality in 40 patients with severe angina or myocardial infarction subjected to urgent surgery.

obtained from the physician who previously attended the patient. These patients were also discarded.

For 40 patients suitable for study, tabulations were made and the following information recorded: (1) age and sex, (2) diagnosis and operation, duration, type of anesthesia, postoperative complications, blood pressure fluctuations, extent and nature of parenteral fluid therapy; (3) cardiac pathology, occurrence, manifestations, and duration, and (4) end-results and significant necropsy findings.

RESULTS

Age and Sex.—As might be anticipated, three-fourths of the patients were in the older age group (60 to 80 years). The youngest was aged 41 years, while the oldest was aged 87. Sixty-six per cent of the patients were men, a ratio of 2 to 1. The preponderance of men corresponds to the prevalence of coronary-disease mortality in that sex. The relationship of age to operative survival rate is shown in Chart 1.

The usual correlation of increased mortality with advancing years is observed. The significance of this fact appears to be emphasized in that group of patients who

died of noncardiac complications. The average age of this group was 74 years, or 12 years more than the average age of survivals. Patients who died of cardiac causes averaged 66 years in age, or 4 years more than the age of those who survived.

DIAGNOSIS AND OPERATION

There were 55 operations performed on 40 patients with myocardial infarction or severe angina pectoris. The primary indications for urgent surgery were the following: (1) to eradicate a malignant tumor, (2) to relieve obstruction of the alimentary or genitourinary tract, and (3) to relieve pain. The principal operations performed in the 40 patients in this study are indicated in Table 1. In one instance

TABLE 1.—*Principal Operations Performed in 40 Patients with Severe Angina Pectoris or Myocardial Infarction*

Operation	No. of Cases	No. of Deaths
Transthoracic vagotomy	1	1
Transthoracic gastroesophagectomy	1	1
Gastrectomy and pancreatectomy	1	1
Exploratory laparotomy	2	0
Cholecystectomy	4	1
Common hepatic duct exploration and/or reconstruction	3	0
Resection of ileum	1	0
Appendectomy	1	1
Colon resection	6	3
Ileotransverse colostomy	1	1
Colostomy	5	4
Abdominoperineal resection	1	1
Hemorrhoidectomy	2	0
Prostatic resection	2	0
Nephrectomy	1	0
Dorsal sympathectomy	4	0
Stellate ganglionectomy	2	0
Lumbar sympathectomy	1	0
Herniorrhaphy	1	0
Radical mastectomy	1	1
Total	41	15

two principal procedures were carried out in the same patient, which accounts for the discrepancy in the additional number of cases tabulated. In approximately three-quarters of the patients (28 cases) the principal operation was on the gastrointestinal tract for malignant or chronic inflammatory disease. Operations on the sympathetic nervous system were performed in seven patients for cardiovascular disease. Three patients were subjected to major genitourinary tract surgery. One patient had a radical mastectomy performed, and another was operated on for an umbilical hernia.

Two or more surgical procedures were performed on 11 patients with known prior myocardial infarction or severe angina. One patient was subjected successfully to surgery on four different occasions. Staged bowel resections and bilateral sympathectomies accounted for the majority of multiple operative procedures. Three patients required secondary closure owing to wound disruption. There were no immediate surgical deaths (Table 1).

The effect of average operating time on the course of 40 patients with severe angina pectoris or myocardial infarction is shown in Chart 2.

The average operating time in those patients who later died of cardiac causes was observed to be actually shorter by 20 minutes than was the case in those who survived. It appears significant that all patients (five cases) who died of noncardiac complications were subjected to the longest operative procedures (2 hours 40 minutes). Four of the five in this group died as a result of an extensive pulmonary infarction. This fact suggests the need for applying prophylactic measures to reduce the hazard of pulmonary embolism in the presence of prolonged surgery.

There appears to be no constant relationship between the volume and type of fluid therapy to mortality. No correlation was observed between mortality and the number of operations, or the complications requiring secondary operative procedures. No deaths attended surgery of the sympathetic nervous system.

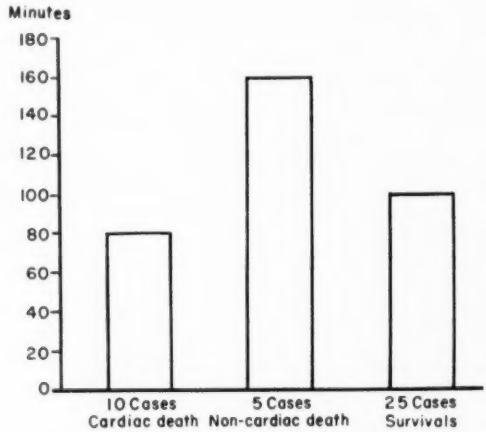

Chart 2.—Duration of surgery and results. Effect of average operating time on the course of 40 patients with myocardial infarction or severe angina.

ANESTHESIA

The anesthetic agents employed and the frequency of their administration are shown in Table 2. Two-thirds of all anesthetics were administered by inhalation, while the remainder were about equally divided between intravenous injections of thiopental (pentothal*) sodium and local injections of procaine hydrochloride. Tribromoethanol (avertin*) was employed only once. Since most patients (90%) were anesthetized with more than one agent, the total number of times anesthesia was used, Table 2, exceeds the total number of procedures, Table 1. There appears to be some reduction in the use of thiopental sodium (45.4%) as a primary or supplementary agent in these cardiac patients. In more than 15,800 administrations of anesthesia to general surgical patients at the Presbyterian Hospital during the last two years, thiopental was employed in more than one-half the instances.[3] The predominant use of this agent in general surgery is apparently experienced in other institutions

3. Anderson, C. D.; deLira, R.; Fisher, D., and Paul, O.: Cardiovascular Complications Encountered During Pentothal* Anesthesia, presented before the Chicago Society of Anesthesiologists and the Chicago Heart Association, Chicago, March 13, 1951.

of comparable size.[4] There appeared to be no constant relationship between mortality and the nature of the anesthetic or the route of administration. The hazard of anesthetic hypoxia associated with thiopental and nitrous oxide is believed to be great, particularly in the presence of myocardial infarction. The following two cases serve to substantiate this impression.

CASE 1.—E. W., a 73-year-old man (blood pressure 130/70), with two previous episodes of myocardial infarction, experienced a sudden drop of pressure (50/0) during a bowel resection. The anesthetic agents used were thiopental, nitrous oxide, and ethylene. Immediately, the surgeon noted the darker color of the blood. A rise in blood pressure and a return of normal color was achieved by the administration of oxygen, but later evidence showed the presence of an undoubted myocardial infarction.

CASE 2.—E. U., a 70-year-old man (blood pressure 198/90), with no previous known myocardial infarction, experienced a drop in blood pressure (130/?) accompanied by cyanosis during the first stage of a combined abdominoperineal resection. The reduction of anesthesia (thiopental and nitrous oxide) and bronchial aspiration brought some improvement (blood pressure 170/80), but cyanosis persisted and evidence of congestive heart failure appeared. The electrocardiogram was interpreted as showing abnormalities characteristic of posterior myocardial infarction and myocardial anoxia. The operation was rapidly and successfully terminated (first stage) with the use of oxygen and intravenous injections of ouabain and with the patient in a slight Trendelenburg position. He subsequently died, and a large myocardial infarct was demonstrated at autopsy.

TABLE 2.—*Anesthetic Agents Employed and Frequency of Administration in 55 Operations*

Anesthetic Agent	No. of Times Used	Cases, %
Inhalation		
Ethylene	26	52.0
Nitrous oxide	25	45.4
Ether	18	32.7
Cyclopropane	4	7.2
Intravenous		
Thiopental (pentothal®) sodium (supplemented or complemented)	25	45.4
Regional		
Procaine hydrochloride	1	1.8
Piperocaine (metycaine®) hydrochloride	2	3.6
Local		
Procaine hydrochloride	14	25.4
Cocaine	1	1.8
Other		
Tribromoethanol (avertin®)	1	1.8
Curare	4	7.2

It is our firm conviction in the light of the above experience and from a recent survey of deaths occurring during anesthesia at the Presbyterian Hospital[3] that the use of thiopental is extremely hazardous in cardiac patients. It is apparent from this survey[3] that the majority of deaths and cardiac arrests during anesthesia may be traced to anesthetic hypoxia, regardless of the anesthetic agent.

CARDIAC PATHOLOGY

Seven patients with severe angina pectoris without myocardial infarction were subjected to surgery. The indication for surgery in six of these cases was status anginosis. There were no deaths.

4. Glazener, E. L., and Hale, D. E.: An Analysis of Anesthesia Administration for 1950, Cleveland Clin. Quart. **19**:40, 1952.

In 33 patients one or more myocardial infarcts developed either before operation, during operation, or in the convalescent period.

A history of myocardial infarction was obtained in two-thirds of these patients (23 cases) on admission, while in one-third (10 cases) there was no evidence of previous myocardial infarction. Patients subjected to surgery with a previous history of myocardial infarction accounted for approximately one-half the mortality (36%) as compared with those who gave no evidence of previous myocardial infarction (63%).

The survival rate of all patients (37%) encountered in this study is shown in Chart 3. It is apparent that patients with multiple infarcts constitute a poorer surgical risk (mortality, 60%) than those patients with a single episode of myocardial infarction (39%).

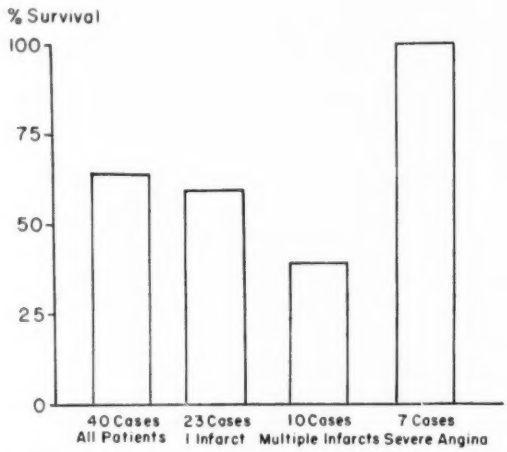

Chart 3.—Survival rate in 40 patients with severe angina and myocardial infarction subjected to urgent surgery.

Operative risk based on the relationship of age of the myocardial infarction to surgery also appears to be significant (Chart 4). The surgical risk in patients with myocardial infarction appears to be related to the duration of infarction prior to operation. Arbitrarily, a critical period of three months was selected because it was believed that this time would be required to complete the essential repair of the damaged wall. There were 20 patients who had evidence of myocardial infarction more than three months before operation. Among this group, one died of a cardiac cause (mortality 5%), another died of a technical error (mediastinitis), while four died of pulmonary embolism. These patients who died of pulmonary embolism were the oldest, with an average age of 74 years, or 12 years more than the average age of survivors. Furthermore, this group of patients experienced the longest operative procedure, averaging one hour more than the survivors.

There were three patients operated on who had a history of myocardial infarction less than three months before operation. Of this group, two died of cardiac causes (mortality, 66%).

Ten patients experienced myocardial infarction during the operative or convalescent period. In each of these instances there was no history of previous existing coronary disease obtained before operation. Seven of these patients died during the convalescent period of cardiac causes.

It is apparent that the risk of cardiac death in patients with known preexisting myocardial infarction is not excessive (5%), whereas the hazard is great in those patients with recent infarction or in those experiencing infarction during the operative or convalescent period.

The majority of myocardial infarctions involved the anterior wall (45%), while in eight instances (36%) the posterior wall was affected. The left lateral wall was involved three times (14%) and the interventricular septum once (5%).

There appeared to be no definite relationship between the patient's admission blood-pressure reading and the survival rate. However, the reduction in diastolic

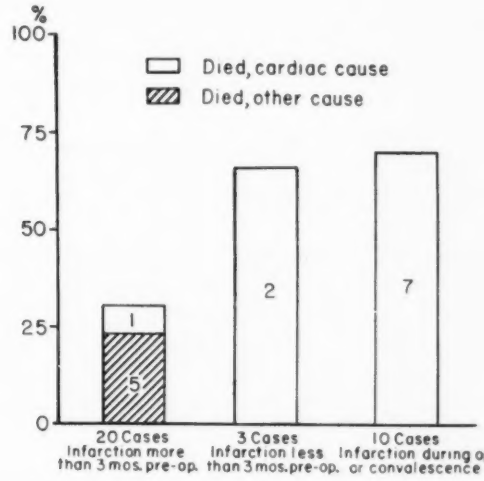

Chart 4.—Surgical risk in myocardial infarction. Relationship of myocardial infarction to surgery and mortality rate.

blood pressure during surgery in those patients who died of cardiac causes was twice as great as that seen in those patients who survived.

There was no correlation of heart size, as determined by chest x-rays, and mortality in patients with previous myocardial infarction and normal blood pressure. A direct relationship was observed, however, between mortality and heart size in patients with both coronary disease and hypertension.

COMMENT

It is apparent to us that the mortality statistics expressed in this study are heavily weighted, since we have surveyed principally those cases of myocardial infarction which were of such magnitude as to produce unmistakable clinical evidence of myocardial disease. None of the 56 patients, with dubious evidence of infarction or angina pectoris, previously excluded from this study died of myocardial infarction.

The high incidence of pulmonary embolism (36%) observed in this series of cardiac patients subjected to surgery is not unusual. It has been pointed out [5] that heart disease is associated with a higher incidence of fatal pulmonary embolism than any other disease. About one-quarter of all cases of thromboembolism occurred in patients with heart disease, while in 40% of the patients studied pulmonary emboli developed. Therefore the need of reducing factors favoring intravascular coagulation is of paramount importance. It is believed that vascular stasis constitutes a major role in the formation of thrombosis. Efforts to increase venous return from the lower extremities by compression bandages and early ambulation and the judicious use of postoperative anticoagulants in cases of prolonged surgery may reduce these complicating fatalities.

CONCLUSIONS

As a result of this study, certain factors influencing mortality are apparent and several recommendations are offered in an effort to reduce the operative risk in patients with myocardial infarction.

1. Patients experiencing recent myocardial infarction (less than three months before operation) should not be subjected to major surgery unless an emergency exists.

2. The prevention of hypoxia during anesthesia is essential, particularly in the presence of known myocardial infarction. The more frequent use of complementary local anesthesia, thus reducing the amount of the primary anesthetic agent, will aid in this objective.

3. Because of the significant association of fatal pulmonary embolism occurring in aged patients with myocardial infarction subjected to prolonged surgery, it is recommended that appropriate measures be instituted to reduce the possibility of intravascular coagulation during the immediate postoperative period.

4. It is recognized that there is no substitute for a careful history and physical examination. However, for all patients more than 45 years of age on whom major surgery is contemplated an electrocardiogram should be made to ascertain the presence of unsuspected coronary disease.

5. Ochsner, A.; DeBakey, M. E.; DeCamp, P. T., and DaRocha, E.: Thrombo-Embolism: An Analysis of Cases at the Charity Hospital in New Orleans over a 12 Year Period, Ann. Surg. **134**:405, 1951.

DUPUYTREN'S CONTRACTURE

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DUPUYTREN'S contracture was first shown to be a disease of the palmar fascia by Dupuytren just 120 years ago. Since that time an exhaustive literature has grown up about it, but knowledge concerning its exact etiology is still not complete, nor is there agreement as to the nature of the pathologic changes which are found in the excised fascia. There is less argument relative to the treatment. It is generally admitted that the condition should be managed surgically; no conservative measures have proved to be satisfactory. There is some argument among surgeons as to how extensive the surgical treatment should be. In general it can be said that most surgeons advise complete removal of the palmar fascia and of prolongations into the fingers if they are involved. Removal should be done fairly early, before secondary changes and extensive skin involvement have taken place. There has been a recrudescence of subcutaneous division of the involved cords, first proposed by Cooper and popularized by Adams. Luck,[1] of Los Angeles, has recently reported on a series of 100 patients treated in whole or part by division of cords by a special fasciotomy knife. Because of the extensive nature of the complete operation some surgeons have advised limited excision, which is much less disabling and can be repeated if further trouble develops. Hamlin [2] has reported 14 patients operated upon by partial excision, in two of whom further partial excisions were required. Both of the latter procedures show higher incidence of "recurrence" than does the complete operation.

ETIOLOGY

It is scarcely necessary to discuss at length the numerous theories which have been advanced concerning the etiology of Dupuytren's contracture. Meyerding and his associates [3] in their several publications on the subject have reviewed very

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From the Department of Surgery, Northwestern University Medical School and Passavant Memorial Hospital.

1. Luck, J. V.: Dupuytren's Contracture: Treatment by Subcutaneous Fasciotomy and Excision of Nodules and Plaques, read at the meeting of the American Academy of Orthopaedic Surgeons, Chicago, Jan. 29, 1952.

2. Hamlin, E. Jr.: Limited Excision for Dupuytren's Contracture, Ann. Surg. **135**:94-97, 1952.

3. Meyerding, H. W.: Dupuytren's Contracture, Arch. Surg. **32**:320-333, 1936. Meyerding, H. W.; Black, J. R., and Broders, A. C.: The Etiology and Pathology of Dupuytren's Contracture, Surg., Gynec. & Obst. **72**:582-590, 1941. Meyerding, H. W., and Jackson, A. E.: Epidermoid Cyst of the Left Palm with Dupuytren's Contracture of the Hands and Right Foot: Case Report, S. Clin. North America **28**:1031-1039, 1948.

well our knowledge concerning the disease. Skoog in a recent monograph has discussed and classified all theories up to 1948 and nothing essentially new has been added since that time. Out of the great bulk of material published, two factors recur with sufficient frequency and backed by enough statistical data to warrant serious consideration. One factor is that of heredity, the other that of chronic trauma.

The hereditary factor is conceded to be significant even by those who are most ardent proponents of a traumatic element in the causation of the disease. Koch [4] has stated that the "most significant fact. . . . with reference to the development of the disease is the frequency with which one is able to elicit a history of similar involvement in other members of the family and in the males particularly." It has been traced through as many as five generations, and in practically every reported series a familial incidence is recorded, often as high as 40%. Couch [5] has reported an instance of identical twins with the contracture. There is no agreement as to whether it is dominant or recessive, and there is some possibility that it may be sex linked.

When we come to consider the significance of trauma in the causation of Dupuytren's contracture, the case is not too clear. A single injury is seldom credited with starting a contracture. In reported instances the relationship is questionable; other conditions are often confused with Dupuytren's, and not infrequently the contracture appears later in the hand not involved in the injury.

The situation for chronic repeated trauma is somewhat different. It is not valid to draw conclusions from any individual instance, nor does any reported series of cases entirely support a chronic traumatic origin. However, studies of large series of patients categorized according to their type of work have shown a higher incidence of the contracture among those persons engaged for long periods of time in hard manual work. Especially does this seem true in those persons whose palms are subject to repeated trauma. However, as has been pointed out by Stuart Gordon,[6] in reported series of patients coming for operative care, over 50% were "non-workers." Certainly the disease can develop without chronic trauma.

Against trauma there are the commonly accepted arguments that the contracture occurs in persons whose hands are not subjected to chronic injury of any sort. The contracture may occur in a hand which is repeatedly traumatized, and appear later in the opposite hand. It is often but not always bilateral, and frequently symmetrical (Fig. 1).

In support of chronic trauma, Skoog has described in his histologic study of the diseased fascia foci of rupture of the collagen fibers. He postulates some condition of the fascia which predisposes it to tiny tears which heal with scar formation and excessive thickening and contracture. However, he recognizes the "fundamental importance of individual predisposition."

4. Koch, S. L.: Dupuytren's Contraction, J. A. M. A. **100**:878-880, 1933.

5. Couch, H.: Identical Dupuytren's Contracture in Identical Twins, Canad. M. A. J. **39**:225-226, 1938.

6. Gordon, S.: Dupuytren's Contracture, Canad. M. A. J. **58**:543-547, 1948.

The occurrence of a contracture of the plantar fascia similar to that in the palmar fascia is well recognized, and, while not nearly so frequent as is Dupuytren's contracture of the hands, is undoubtedly of the same nature. Meyerding and Shellito reported an incidence of 23 cases in 800 instances of Dupuytren's contracture. Knuckle pads are also frequently associated with Dupuytren's contracture and appear to be part of the picture. As to Peyronie's disease, the relationship is not so clear. The incidence of Dupuytren's contracture among patients with Peyronie's disease seems to be higher than does the reverse relationship. Scott and Scardino[7] found 6 instances of Dupuytren's contracture in 23 cases of Peyronie's disease. Certainly my experience with several hundred patients with Dupuytren's contracture would hardly yield two with Peyronie's. In this connection, however, it must be said that my attention has been directed only lately to the possible association of the two conditions.

Fig. 1.—Bilateral and symmetrical Dupuytren's contracture. The right hand was operated upon in March, 1933; the left hand in March, 1936. A free full thickness skin graft was required in the left palm. The photographs in the lower row show the hands in December, 1951.

SYMPTOMS AND SIGNS

The symptoms and findings of Dupuytren's contracture are such that a detailed description is out of place at the moment. It begins as a nodule in the palm, usually in the region of the distal palmar crease, as a rule in a line with the ring finger. It may remain stationary for months or years, causing few symptoms, or it may be followed by the development of a fibrous cord in the palm proximal to the nodule, or distal to it, or both. As the cord develops it tends to pull the finger distal to it into flexion at the metacarpophalangeal joint, and later at the proximal

7. Scott, W. W., and Scardino, P.: A New Concept in the Treatment of Peyronie's Disease, South. M. J. **41**:173-177, 1948.

interphalangeal joint. The ring, little, and middle fingers are usually involved in that order, singly or together; more rarely is the index finger or the thumb affected. The palmar mass may consist of distinct bands, or it may take the form of a broad dense plaque, intimately fused with the overlying skin, which is thrown into folds and pits over it. In the fingers there may be distinct bands, or the whole volar surface of the proximal phalanx may be occupied by a hard, thick, lozenge-shaped mass.

Subjective symptoms are usually not marked. There may be some numbness and mild pains at the onset, and the nodules may be painful to pressure and blows. I have seen one patient who complained of severe or rather continuous pain and distress, which were much relieved by excision of the fascia. Careful study failed to reveal any other cause for the distress. Bolgert [8] mentions the hyperalgesic type of Dupuytren's contracture characterized by intense night pain and insomnia. It must be more rare in this country than in France.

OPERATIVE TREATMENT

The surgeon cannot predict the course which a given contracture will take. The nodule may cause few or no symptoms, and if no contracting bands develop the patient may not be disabled in any way by the disease. Operative intervention can scarcely be urged in this stage. However, if disability is present or if the process is growing, it is advisable to operate. As the condition develops the overlying skin becomes more and more involved in the process and the probability of having to excise skin becomes more likely. It is my feeling that if operation is undertaken it should consist in complete removal of the fascia. Only in exceptional cases would it seem that partial procedures are indicated. Subcutaneous fasciotomy seems to me to be a hazardous operation in view of the intricate anatomic relationship of the contracted fascia to nerves and blood vessels in the distal palm and fingers. The incidence of recurrence is likewise higher with subcutaneous fasciotomy.

Complete removal of the fascia entails excision of not only the palmar plate but also the deep vertical sheets which pass from the under surface of the fascia to the metacarpals. These sheets, which are intimately associated with the neurovascular bundles, must be excised with care.

The operation is done usually under general anesthesia and always in a bloodless field, secured with a sphygmomanometer, since the surgeon must clearly visualize nerves and blood vessels at all times. Operation under local anesthesia is not satisfactory if a complete excision is attempted. Langston and Badre [9] recommend brachial plexus block, which we have not used. In the region of the metacarpal heads, where the fascia runs distally toward the finger, the neurovascular bundles are enclosed between two layers of fascia which can be traced into the proximal phalanx. These fascial layers often contract and in so doing distort the nerves and vessels in

8. Bolgert, M.: Maladie de Dupuytren: Conssinets des phalanges, Semaine hôp. Paris, **26**:3011-3017, 1950.

9. Langston, R. G., and Badre, E. J.: Dupuytren's Contracture, Canad. M. A. J. **58**:57-61 1948.

their course. The dissection at this region in the palm and over the proximal phalanx of the finger must be done with particular care. The nerves must be visualized at all times to avoid injury.

Incisions for exposure follow the physiologic lines for hand incisions, as is emphasized by Gordon. Transverse incision across the palm at the level of the distal palmar crease will permit exposure of the whole of the palmar plate of fascia from the region of the webs upwards to the level of the superficial volar arch. Usually a second incision about 2 or 2½ in. (5 or 6 cm.) long, skirting the thenar eminence, is needed to expose the proximal end of the palmar plate.

Great care is exercised in separating the fascia from the overlying skin. It is important to save the skin if possible; it is surprisingly viable and recuperative and can often be dissected free even when badly involved. I cannot go quite so far as Buff [10] in stating that the skin should never be excised, but certainly every effort should be made to save it. However, there are cases in which it is not possible to save the skin, and here the surgeon must excise and replace. In making the excision the surgeon plans it so that the suture lines of the free graft do not lead to contracting scars.

When the overlying skin has been carefuly dissected free, the fascia may be attacked. Starting proximally the surgeon divides the attachment to the transverse carpal ligament and then incises the fascia laterally along the border of the thenar eminence and medially along the hypothenar eminence. The whole plate may then be drawn distally with only a few deep attachments until just distal to the superficial volar arch where the deep sheets are met with. From this point on (Fig. 2) it is helpful to divide the pretendinous bands longitudinally and come down directly upon the underlying tendon. The neurovascular bundles lying on either side of the tendon enclosed in the deep sheets of the palmar fascia are then visualized. These deep sheets are then divided close to their attachment to the deep palmar fascia, nerves and vessels being kept constantly in view. A small spatula used by an assistant as a retractor is very helpful at this stage of the dissection.

Removal of fascia from the volar surface of the proximal phalanges of the fingers may be accomplished through a midlateral incision with a cross incision near the interphalangeal joint. I prefer, however, an oblique incision over the proximal phalanx, which permits exposure of nerves to either side and allows dissection of the fascia from the under surface of the flaps. The oblique incision is especially valuable when the digital involvement takes the shape of a plaque. When the skin on the volar surface of the digit is contracted badly, a Z-plasty incision, as recommended by McIndoe (cited by Skoog), is very advantageous. Iselin,[11] in a recent report covering his experiences over the past 20 or more years, recounts the experiences with various operative technics. This surgeon has recently adopted what he terms a total Z-plasty, not only for the fingers but for the palm as well.

Careful hemostasis is essential to success of the operation. The surgeon will be amply repaid for the extra time it takes to clamp and ligate bleeders as he goes along

10. Buff, H. U.: Bemerkungen zur Therapie der Dupuytrenschen Kontraktur, Helvet. chir. acta **17**:277-280, 1950.

11. Iselin, M., and Dieckmann, G.: Notre expérience du traitement de la maladie de Dupuytren, Mém. Acad. chir. **77**:251-255, 1951.

and for spending time before closure in ligating vessels which show up after the cuff has been released. I have also found it helpful to tie a compression dressing over the palm after closure. This may be done by means of silk sutures which are introduced into the skin on either border of the palm and tied over a large mass of fluffed gauze. This fluffed mass should be removed in four or five days, since the overtie sutures tend to cut through the skin if left in too long.

In some instances it will be necessary to excise skin which is severely involved in the contracture. The defects thus left may be closed by free full-thickness skin grafts, carefully sutured in place and held against the bed with silk overties. Bruner [12] has described a dorsal rotation flap, swung in from the ulnar side into the palm. The bed of the rotation flap is covered by a split graft.

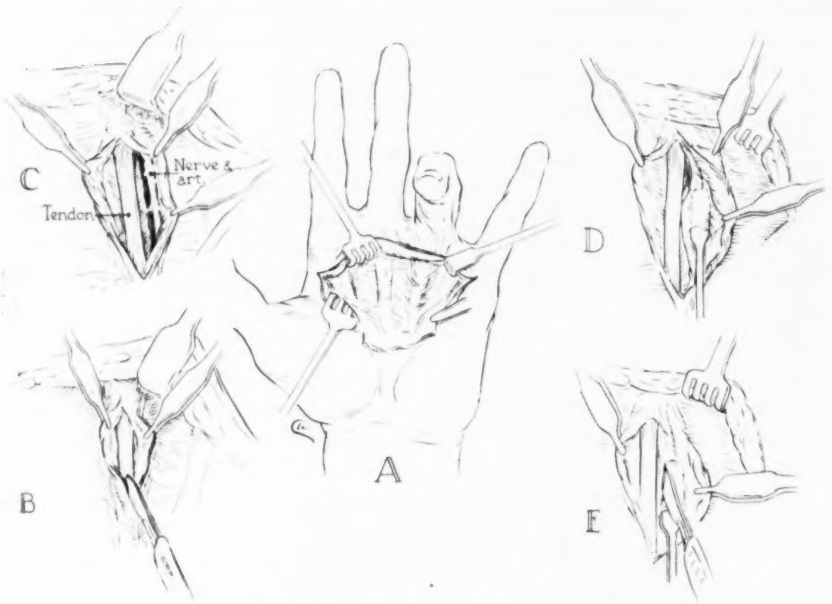

Fig. 2.—The palmar fascia is uncovered through a transverse incision in the palm and usually by a second incision skirting the thenar eminence (*A*). The base of the fascia is divided where it takes origin from the transverse carpal ligament and drawn distally. Distal to the superficial volar arch, the deep vertical sheets of fascia hold the palmar plate securely attached to the deep palmar fascia. These sheets likewise contain the neurovascular bundles. In order to visualize these sheets the pretendinous bands are divided longitudinally so as to come down upon the underlying tendons (*B*). When the two halves of the band are drawn aside (*C*), the nerves and vessels may usually be visualized, covered by a layer of fascia. A spatula used as a retractor is then introduced beneath the vertical sheet, and the surgeon may then divide this layer close to its attachment to the deep palmar fascia (*D* and *E*). By following cautiously along distally the surgeon can keep the nerves and vessels under constant visual control.

Following operation the hand is dressed in a compression dressing on a dorsal splint leaving the distal phalanges of the fingers free and not incorporating the thumb.

12. Bruner, J. M.: The Use of Dorsal Skin Flap for the Coverage of Palmar Defects After Aponeurectomy for Dupuytren's Contracture, Plast. & Reconstruct. Surg. **4**:559-565, 1949.

Splinting is done in a slightly flexed position so as not to put tension on the palmar suture line. The initial dressing on the fourth or fifth day is done to divide the silk overties and to remove tiny rubber strip drains that were introduced into the palmar suture line. At the next dressing, three or four days later, part of the sutures are removed and the remainder a few days later.

Compression dressing and splint are left on for about 10 to 14 days, when, if healing is progressing satisfactorily, the splint is removed to permit the patient to start moving the fingers. As soon as healing is complete, the patient is instructed to wash the hand gently once or twice daily in warm soapy water. Use is permitted, but violent massage or violent stretching is forbidden. Stiffness will be present and it will take some time to work it out, but the patient is cautioned that forceful stretching and energetic physical therapy will lead to tears, increase scar tissue formation, and prolong healing.

DISCUSSION

Dr. Minot P. Fryer, St. Louis: I wonder if Dr. Mason would answer two additional questions that occasionally arise in connection with the treatment of this condition.

First, would it be of any advantage to do a staged, but complete, excision of the palmar fascia in those patients whom you expect will have trouble with the finger joints later on, thereby allowing for earlier mobilization of the fingers?

Second, what are Dr. Mason's recommendations for the treatment of those joints that have been pulled into subluxation removal of the palmar fascia?

Dr. B. Noland Carter, Cincinnati: I should like to ask Dr. Mason what results have been accomplished by vitamin therapy in patients with Dupuytren's contracture?

Dr. Michael Mason, Chicago: I have had no experience with the stage excision, except that once in a while when the condition recurs we do a stage excision and remove the palmar fascia a second time. This does not occur very often, but, of course, it does happen.

Subluxation of the joints occurs particularly in the little finger. This difficulty arises because the fascial bands on the finger do not run on the volar surface only but also along the side of the finger. In such cases if we dissect well along the side we can very frequently free those bands and often improve the situation.

We always have felt that doing the complete operation is preferable to subjecting a hand to two or three procedures if it can be avoided.

Vitamin E has been recommended for Dupuytren's contracture. It was first used by the urologists in the treatment of Peyronie's disease and then was transferred to Dupuytren's contracture. A number of series of cases have been studied. I think the general opinion to date is that vitamin E is not of any real help in the treatment of Dupuytren's contracture.

EVALUATION OF THREE METHODS FOR FINGER TIP INJURIES

ROBERT H. CLIFFORD, M.D.
DETROIT

IN THE highly mechanized industry of today amputation of the finger tip is a relatively frequent injury. In its physical extent it is a minor injury, but for the patient it is rather serious. This disability usually prevents him from performing his regular job for a month or more, occasionally forces him to change his job, and frequently leaves him with a painful and useless finger stump.

The literature on this subject is rather meager.[1] Most of the articles discuss and describe the three main types of treatment, but almost none of them evaluate the results. In most of the articles the statement is made, without qualification, that length must be preserved at all costs.

Because of its nobility of purpose, this statement is rarely challenged. It should be challenged, however, for the preservation of a centimeter or two of finger length which may terminate in a painful and useless tip is hardly a triumph of therapy.

The three main types of treatment for this injury are free skin graft to the stump, pedicle graft to the stump, and revision, with or without shortening, and primary closure.

To evaluate the results properly it was felt that examination should be done at least two years after operation. The histories of all those treated for amputation of the finger tip beyond the distal interphalangeal joint at Henry Ford Hospital between 1945 and 1949 were reviewed. Of 163 patients, 125 returned for examination. These men and women were examined, and they were questioned about their work records, subjective disability, and persistent symptoms.

From the Henry Ford Hospital.

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

1. Gillies, H.: Autograft of Amputated Digit: Suggested Operation, Lancet **1**:1002-1003 (June) 1940. Gordon, S.: Autograft of Amputated Thumb, ibid. **2**:823 (Dec.) 1944. Horn, J. S.: The Use of Full Thickness Hand Skin Flaps in the Reconstruction of Injured Fingers, Plast. & Reconstruct. Surg. **7**:463-481 (June) 1951. Jones, R. A.: Method for Closing Traumatic Defect of Finger Tip, Am. J. Surg. **55**:326-338 (Feb.) 1942. Kallio, K. E.: Plastic Operations on Thumb, Acta chir. scandinav. **93**:231-253 (April) 1946. McCarroll, H. R.: Immediate Application of Free Full-Thickness Skin Graft for Traumatic Amputation of Finger, J. Bone & Joint Surg. **26**:489-494 (July) 1944. Murray, A. R.: Reconstructive Surgery of Hand, with Special Reference to Digital Transplantation, Brit. J. Surg. **34**:131-140 (Oct.) 1946. Read, F. L., and Haslam, E. T.: Immediate Skin Grafts on Finger Injuries, U. S. Nav. M. Bull. **42**:183-186 (Jan.) 1944. Webster, G. V.: Treatment of Finger Tip Amputation, Postgrad. Med. **8**:416-419 (Nov.) 1950. Zadik, F. R.: Immediate Skin Grafting for Traumatic Amputation of Finger-Tips, Lancet **1**:335-336 (March) 1943.

The number of patients treated by each method are shown in the accompanying table.

Table 2 shows the average disability in weeks before patients could return to their regular jobs. This does not always mean time lost from work. In the large automobile plants in Detroit, from which most of these patients come, they are frequently put back to work at one-handed jobs or other temporary jobs until they are well enough to do their regular work.

Here we see that the simplest method, revision and closure affords a disability of one month, whereas free grafting is disabling for almost two months, and pedicle grafting for about three months.

TABLE 1.—*Types of Treatment in Patients Followed for at Least Two Years*

	No. of Patients	% of Group
Pedicle graft	9.0	7.2
Free graft	34.0	27.2
Revision, shortening, and closure	82.0	65.6
Total	125.0	

TABLE 2.—*Average Weeks of Disability to Perform Regular Job*

	Wk.
Pedicle grafts	12.0
Free grafts	7.2
Revision and closure	4.2

TABLE 3.—*Patients Forced to Change Jobs Because of Finger Tip Injury*

	No. of Patients	%
Pedicle graft	4	44
Free graft	10	29
Revision and closure	8	9.9

Table 3 shows the number of patients who, after the injury had healed and a reasonable trial period was over, were no longer able to carry out the same duties as before injury.

The percentage of patients forced to change their jobs is greatest in the group treated with a pedicle graft, next in the free skin graft group, and least in the revision and closure group.

The subjective complaints of those patients unsatisfied with the results are shown in Table 4.

In those patients with a pedicle graft, the main complaint was lack of sensation and lack of tissue tone. Two of these patients were so dissatisfied that they had the tips amputated.

In those treated by free graft and by revision and closure the chief complaint was the painful stump.

In the successful use of the free graft or of the pedicle graft the results are truly excellent, but the number of completely successful results are somewhat disappointing.

TABLE 4.—*Unsatisfied Patients: Subjective Complaints*

Pedicle graft	6	(66%)
Reamputation	2	
Tender stump	1	
Lack of sensitivity	1	
Lack of tone	3	
Free graft	26	(76%)
Mild to moderate tenderness	14	
Extreme tenderness	12	
Revision	17	(13%)
Persistent nail stump	2	
Moderate tenderness	8	
Extreme tenderness	6	

The large numbers of successful primary repairs, with or without shortening, coupled with the great adaptability of the hand suggest that this method is probably best for the average working man.

TREATED DERMIS AS A SOURCE OF REPARATIVE TISSUE

STUART GORDON
TORONTO, CANADA

FOR NEARLY a century surgeons have sought for a safe simple method of utilizing fibrous tissue in the repair of subcutaneous defects. Kirschner,[1] Neuhof,[2] and Gallie and LeMesurier [3] published reports on the use of fascia lata; others, notably Loewe,[4] Rehn,[5] Uihlein,[6] Mair,[7] Cannady,[8] Peer and Paddock,[9] Harkins,[10] and Marsden,[11] published experimental and clinical data on the use of whole-thickness skin, or dermis. The final answer to the clinical use of skin as a reparative tissue would appear to be contained in Strahan's paper.[12] He reported

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

1. Kirschner, M.: Der gegenwärtige Stand und die nächsten Aussichten der autoplastischen, freien Fascien- Übertragung, Beitr. klin. Chir. **86**:5-149, 1913.

2. Neuhof, H.: Fascia Transplantation, Surg., Gynec. & Obst. **24**:383-427 (April) 1917; Fascia Transplantation in Defects of the Major Arteries, ibid. **26**:324-334 (March) 1918.

3. Gallie, W. E., and LeMesurier, A. B.: The Transplantation of the Fibrous Tissues in the Repair of Anatomical Defects, Brit. J. Surg. **12**:289-320 (Oct.) 1924.

4. Loewe, O.: Über Hautimplantation an Stelle der freien Faszienplastik, München. med. Wchnschr. **60**:1320, 1913; cited by Uihlein, A., Jr.: Use of the Cutis Graft in Plastic Operations, Arch. Surg. **38**:118-130 (Jan.) 1939.

5. Rehn, E., and Miyanchi: Das cutane und subcutane Bindegewebe in veränderter Funktion, Arch. klin. Chir. **105**:1-46, 1914.

6. Uihlein, A., Jr.: Use of the Cutis Graft in Plastic Operations, Arch. Surg. **38**:118-130 (Jan.) 1939.

7. Mair, G. B.: Use of Whole Skin Grafts as a Substitute for Fascial Sutures in the Treatment of Hernias: Preliminary Report, Am. J. Surg. **69**:352-365 (Sept.) 1945; Preliminary Report on the Use of Whole Skin-Grafts as a Substitute for Fascial Sutures in the Treatment of Herniæ, Brit. J. Surg. **32**:381-385 (Jan.) 1945; Analysis of a Series of 454 Inguinal Herniæ, with Special Reference to Morbidity and Recurrence After the Whole Skin-Graft Method, ibid. **34**:42-48 (July) 1946.

8. Cannady, J. E.: The Use of the Cutis Graft in the Repair of Certain Types of Incisional Herniae and Other Conditions, Ann. Surg. **115**:775-781 (May) 1942; Some of the Uses of the Cutis Graft in Surgery, Am. J. Surg. **59**:409-419 (Feb.) 1943.

9. Peer, L. A., and Paddock, R.: Histologic Studies on Fate of Deeply Implanted Dermal Grafts: Observations on Sections of Implants Buried from One Week to One Year, Arch. Surg. **34**:268-290 (Feb.) 1937.

10. Harkins, H. N.: Cutis Grafts: Clinical and Experimental Studies on Their Use as Reinforcing Patch in Repair of Large Ventral and Incisional Herniae, Ann. Surg. **122**:996-1015 (Dec.) 1945.

11. Marsden, C. M.: Whole Skin-Graft Repair of Inguinal Hernia, Brit. J. Surg. **35**:390-394 (April) 1948.

12. Strahan, A. W. B.: Hernial Repair by Whole-Skin Graft, Brit. J. Surg. **38**:276-284 (Jan.) 1951.

413 herniae in which buried skin was used as a reinforcing tissue. A substantial number of postoperative cysts and chronic sinuses due to persistent epithelial cells occurred without an offsetting reduction in the recurrence rate.

That cysts would probably be formed if living epithelial cells were buried in mesothelial tissues was pointed out in 1949,[13] and again in 1951.[14] Such a suggestion was by no means original, but experimental evidence was given to support this view. It did seem rather odd that, although aware of the occurrence of inclusion cysts, surgeons would accept the use of buried skin in the repair of tissue defects.

There should be some method of removing the remaining epithelial cells from a sheet of dermis, leaving an elastic sheet of intercellular substance, collagen, and elastic fibers. Two methods of eliminating the epithelial cells were considered, radiation and rapid freezing. The latter method was employed, and with use of dry ice, strips of dermis were frozen and then thawed three times before they were used. Evidence will be presented indicating that the three rapid freezings do destroy the epithelial cells and thus remove the danger of cyst formation. This tissue will be termed "treated dermis."

Since the publication of our previous experimental work, additional studies have been made both in animals and in man.

EXPERIMENTAL DATA

Areas of peritoneum approximately 1 by 2 in. (2.5 by 5.0 cm.) in size were excised in 15 rabbits and the defects repaired with a patch of treated dermis. These patches were examined at intervals up to eight months. In no instance was a cyst found, nor did a sinus develop. Two patches examined at 11 and 14 days showed, between them, three filmy adhesions to the intestines, the others none. Grossly the peritoneal surfaces resembled the smooth shiny lining of the remainder of the abdominal cavity. Had it not been for the silk suture outlining the patches it would have been difficult to identify the transplant in the living animal (Fig. 1).

Microscopic studies revealed that the peritoneal surfaces of these patches became covered with mesothelium between the 14th and the 21st day. The transplant was replaced by connective tissue, which invaded the graft, laid down in wavy lines parallel to each other and, in the main, to the mesothelial layer. Occasionally, capillaries containing blood could be seen in the transplant as early as one week after transplantation (Fig. 2).

CLINICAL DATA

During the spring of 1948, seven inguinal herniae and one ventral hernia were repaired. Treated dermis was used as a patch to reinforce the posterior wall of the inguinal canal in the former and as a many-tailed sheet in the latter. To date neither a recognizable cyst nor a sinus has developed. No recurrence has been reported of the inguinal herniae, although on four no report has been made since 1949. A slight bulging was apparent in the ventral hernia six months after operation and was unchanged in 1951.

13. Gordon, S.: The Subcutaneous Transplantation of Skin, Brit. J. Plast. Surg. **1**:212-216 (Oct.) 1948.

14. Gray, D. B.; Mansberger, A. R., Jr., and Yeager, G. H.: The Fate of Buried Full-Thickness Skin, Ann. Surg. **134**:205-209 (Aug.) 1951.

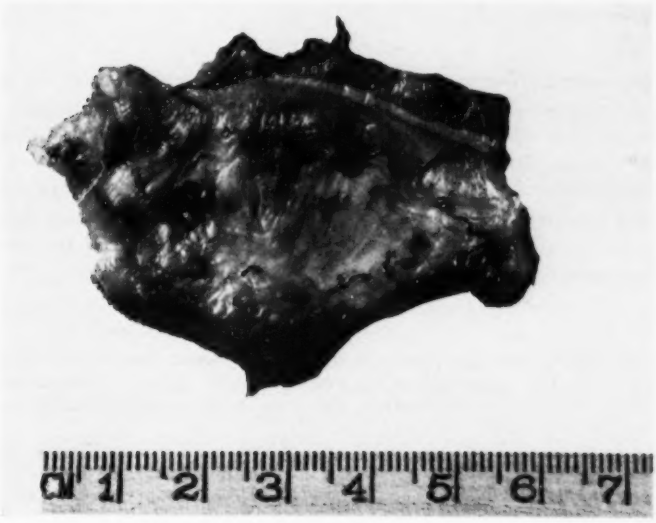

Fig. 1.—Peritoneal patch of treated dermis that had been in place for seven months is outlined by silk suture.

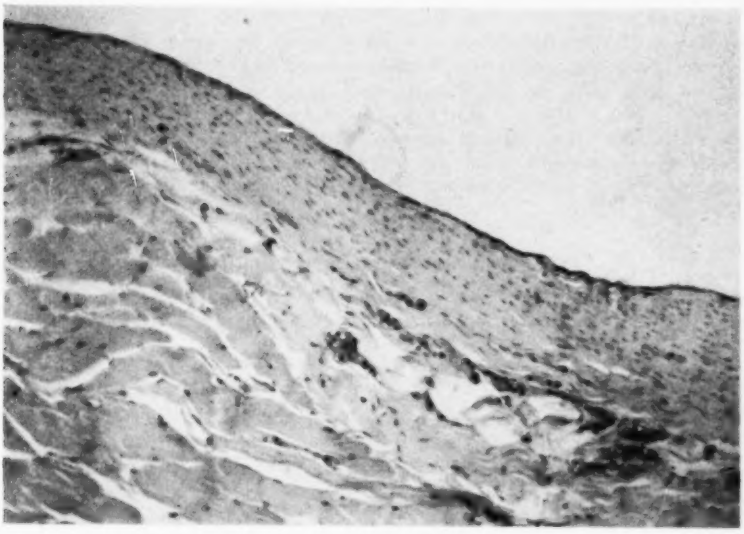

Fig. 2.—Photomicrograph of peritoneal patch of treated dermis removed after eight months.

In the last four years treated dermis has been used to fill in hollows in the forehead, cheek, or nose in eight patients. No complication attributable to the use of the tissue has been noted.

CONCLUSIONS

Autogenous treated dermis appears to be a satisfactory substance for repairing certain types of defects. Experimental work and clinical experience indicate that the danger of cyst and sinus formation has been removed, that sound healing occurs because the transplant is replaced by living connective tissue, and that for a time, at least, treated dermis remains elastic. This last fact suggests that treated dermis would be the ideal supportive tissue to wrap about aneurysms or about suture lines in arteries or veins, sites where a tissue that permitted expansion and contraction would be advantageous.

DISCUSSION

DR. F. G. KERGIN, Toronto, Canada: I have been watching Dr. Gordon's clinical work with dermis, and it seems to me that he has solved a problem. He now offers material which will not give rise to sinus or cyst formation, and at the same time it has retained its essential qualities.

In our general surgical service we do not often wish to use transplanted tissue of this type as a patch in repairing a hernia. Generally, we feel that an actual suture is the treatment of choice in even the large ventral hernias. Fascia is very suitable for this, and we have had long experience with it.

However, Dr. Gordon's demonstration of dermis as a patch is valuable because fascia is not a good patch. It is strong in only one direction, whereas dermis is very strong in all directions. My experience with dermis is confined to thoracic work. On two occasions I have repaired a bronchial defect, using the method of Gebauer, with use of a dermis graft reinforced by wire, and it was very satisfactory.

DR. CHARLES G. JOHNSON, Detroit: I should like to ask Dr. Gordon if he has attempted to preserve this material by freezing or by lyophilizing to get rid of the subsequent cyst formation.

DR. STUART GORDON, Toronto, Canada: We have not attempted to save this material or store it. We have not tried any other way of getting rid of the epithelial element. I think there is a possibility that the material might be of value as a homograft.

All our experimental work to date has been with autografts. We are presently carrying on further work in which we are attempting to use it as a homograft.

RECTUS SHEATH GRAFTS IN VASCULAR REPAIR

JAMES E. COUSAR III, M.D.
AND
CONRAD R. LAM, M.D.
DETROIT

THERE is general agreement that if a defect exists in an essential blood vessel, it is best repaired with a piece of vessel from the same individual (autograft) or from an individual of the same species (homograft). The usefulness of homografts in vascular surgery has been amply demonstrated,[1] but there are problems of procurement, preparation, sterilization, and preservation. It would be advantageous if it could be shown that a tissue other than vascular tissue could substitute for vessel wall. It occurred to us that a tough structure like the posterior rectus sheath with its peritoneal lining might have some possibilities. A review of the literature turned up the fact that Alexis Carrel [2] once used a flap of peritoneum to repair a defect in the aorta of a cat, and three months later the vessel appeared to be patent. Recently, Sako and co-workers [3] carried out some experiments with tubes made of pericardium. In a brief preliminary communication they reported that after three months such grafts frequently showed a breakdown of the luminal surfaces and cholesterol deposits.

METHODS

Experiments were carried out on 30 mongrel dogs. The animals were anesthetized with an oxygen-ether mixture through an endotracheal tube. The abdomen was shaved, surgically prepared, and entered through a low transverse incision. A piece of posterior rectus sheath, with attached peritoneum of a size sufficient for the intended repair, was cut off and placed in isotonic sodium chloride solution. The parietal peritoneum overlying the aorta was divided, and the vessel was mobilized from the renal to the iliac arteries. In this location the aorta can be clamped for long periods without producing ischemic residuals.

From the Division of General Surgery of the Henry Ford Hospital.

This work was supported by a grant from the Michigan Heart Association.

Read at the Ninth Annual Meeting of the Central Surgical Association, Toronto, Canada, March 7, 1952.

1. Gross, R. E.; Hurwitt, E. S.; Bill, A. H., Jr., and Peirce, E. C., II.: Preliminary Observations on the Use of Human Arterial Grafts in the Treatment of Certain Cardiovascular Defects, New England J. Med. **239**:578, 1948. Swan, H.; Maaske, C.; Johnson, M., and Grover, R.: Arterial Homografts: II. Resection of Thoracic Aortic Aneurysm Using a Stored Human Arterial Transplant, Arch. Surg. **61**:732, 1950. Lam, C. R., and Aram, H. H.: Resection of the Descending Thoracic Aorta for Aneurysm: A Report of the Use of a Homograft in a Case and an Experimental Study, Ann. Surg. **134**:743, 1951.

2. Carrel, A.: Surgery of Blood Vessels, Bull. Johns Hopkins Hosp. **18**:181, 1907.

3. Sako, Y.; Clatworthy, H. W.; Chisholm, T., and Varco, R. L.: Observations on the Fate of Pericardial and Fascial Tissue Transplanted into the Thoracic Aorta; abstracted in Surgical Forum, Proceedings of the Clinical Congress of the American College of Surgeons, 1950, edited by O. Wangensteen, Philadelphia, W. B. Saunders Company, 1951, p. 310.

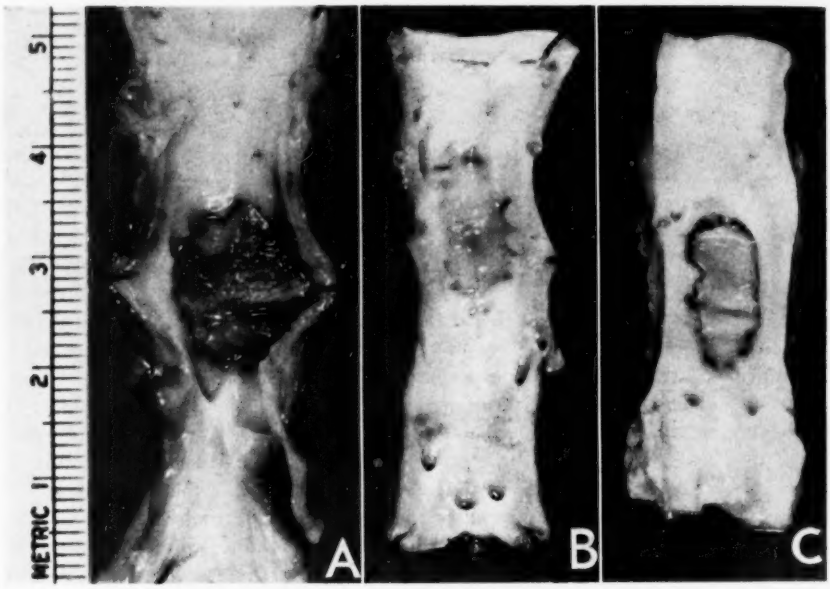

Fig. 1.—Patch grafts of rectus sheath and peritoneum implanted for the following periods: *A*, five days; *B*, three months; *C*, one year.

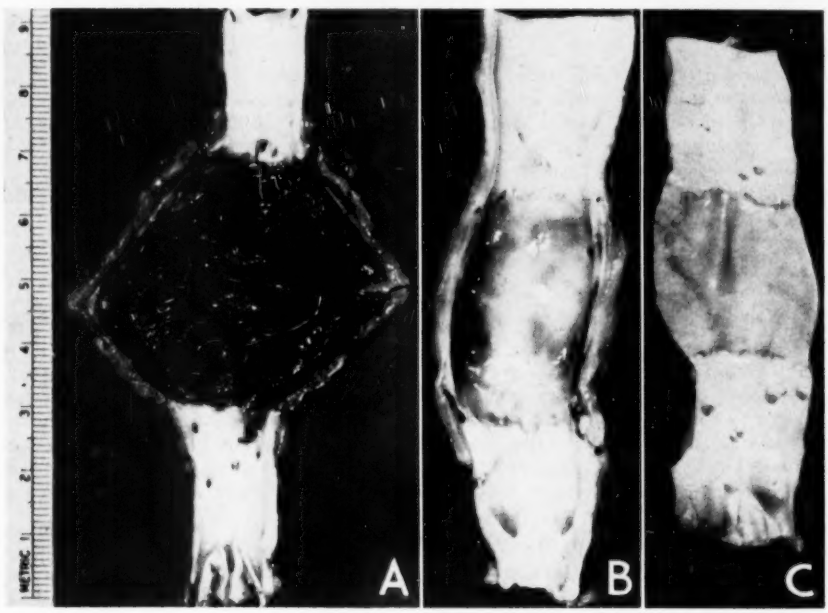

Fig. 2.—Tubular grafts of rectus sheath and peritoneum implanted for the following periods: *A*, five days; *B*, two months; *C*, ten months.

Two types of defects were made in the aortas and appropriately repaired with rectus sheath grafts. For the first type, an ovoid section of the anterior aortic wall was removed; the width of the defect was approximately half the circumference of the aorta. The second type of defect was an interruption of aortic continuity by the excision of a full circumferential segment, 2 to 3 cm. in length. For bridging these gaps, rectus sheath tubes were constructed by placing the material around a syringe plunger of suitable size and approximating the lateral edges with two running sutures of 5-0 Deknatel braided silk. In the earlier experiments the graft to aorta suture lines were placed through a single layer of rectus sheath and peritoneum, but the incidence of breakdown of these suture lines led us to turn back a cuff at each end of the tube to increase the thickness. In placing the grafts, both patches and tubes, we used a continuous horizontal mattress suture of 5-0 Deknatel silk. Anticoagulants were not used.

The animals that died in the immediate postoperative period were studied at autopsy promptly; others were killed at intervals of from five days to one year. The graft specimens were photographed, and histologic sections were made of many of them. An aortogram (iodopyracet [diodrast®]) was made on one animal.

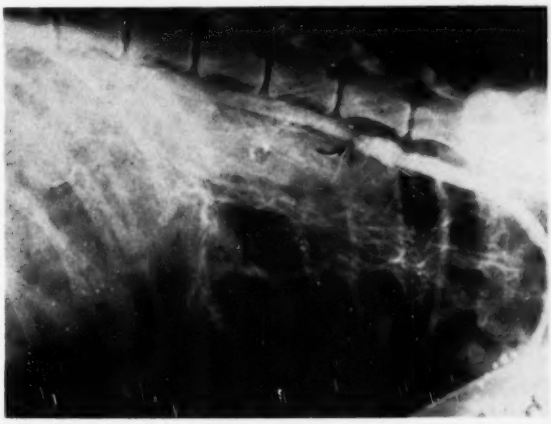

Fig. 3.—Aortogram showing tubular graft after one year. Arrows indicate suture lines, where there is slight constriction.

RESULTS

Rectus sheath patches were inserted into the aortas of 15 dogs. One animal is still living 15 months after operation and has always had good pulsations in both femoral arteries. In two of these animals, leg paralysis developed during the first few days, and they were killed. In both animals thrombosis was present in the region of the grafts. There was considered to be a serious technical error in one, since constriction of the aorta had resulted from the manner in which the suturing was done. The appearance of the grafts in the other 12 that were killed will be described later.

Tubular grafts were placed in 15 animals. One animal was still under observation 14 months after operation; there were good pulsations in the femoral arteries. Of the other 14, it is known that there were 6 failures and 8 good results. The failures were due to hemorrhage and thrombosis in two, hemorrhage alone in two, and thrombosis alone in two. Five of the failures were evident within the first postoperative week. There was only one instance of breakdown of the suture line in five animals in which tubes with cuffs turned back were used.

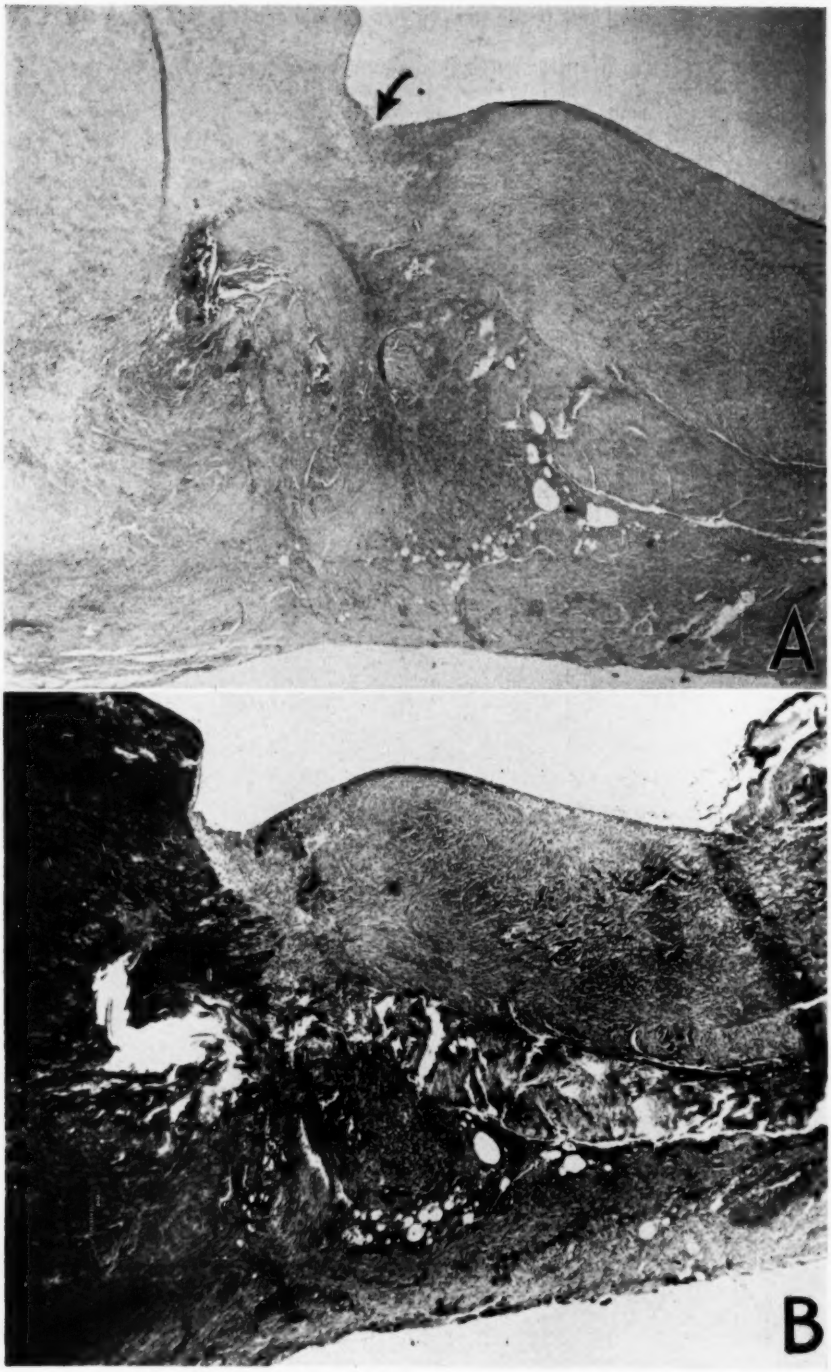

Fig. 4.—Photomicrographs of specimen removed 15 days after operation. *A*, arrow indicates anastomosis rectus sheath graft on the right; hematoxylin and eosin; low power. *B*, rectus sheath graft on the right; elastica trichrome stain; low power.

Four dogs with patches and three with tubes were killed between 5 and 15 days postoperatively for the purpose of studying the early changes in the grafts. The earliest specimens show a marked reaction on the peritoneal or "intimal" sur-

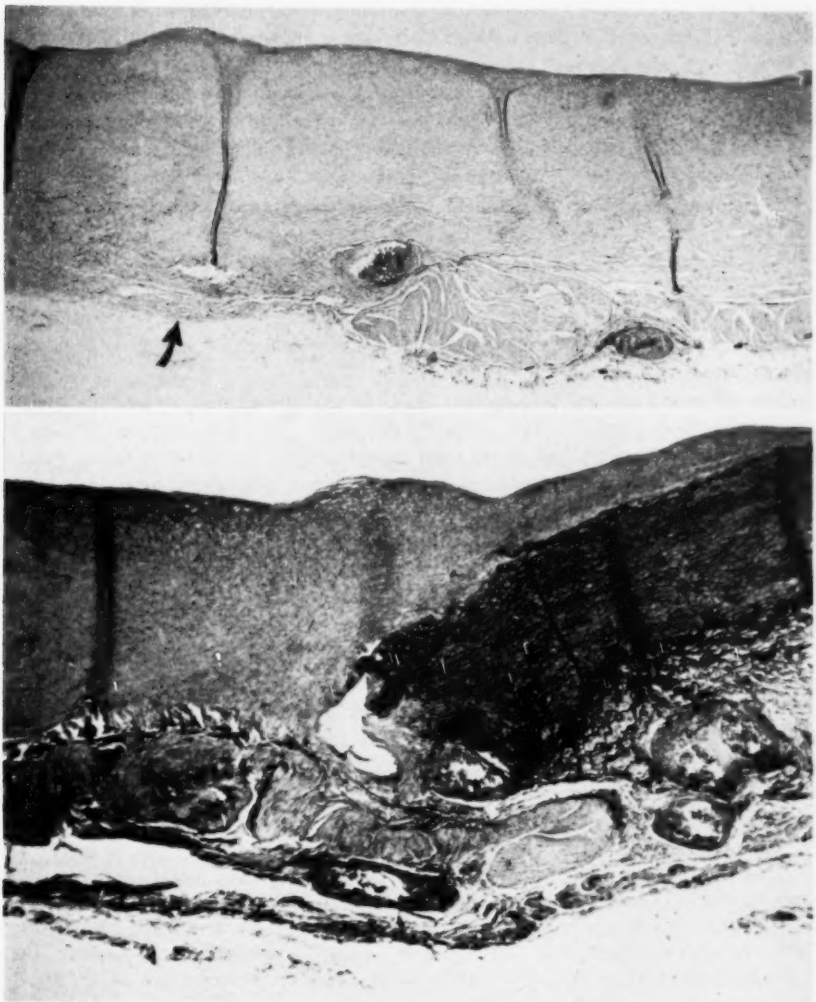

Fig. 5.—Photomicrographs of each end of specimen removed six months after operation. *A*, rectus sheath graft to right of arrow; hematoxylin and eosin; low power. *B*, rectus sheath graft on left side of photomicrograph; elastica trichrome stain; low power.

faces (Figs. 1*A* and 2*A*). A reddish coagulum appeared to cover the surfaces; the grafts were irregularly thickened. Later specimens from this group showed a marked clearing of the coagulum, and a glistening white intima-like surface became apparent over most of the grafts.

An intermediate group of survivors, killed from one to six months postoperatively, included three dogs with patches and two with tubes. Inspection of the specimens (Figs. 1*B* and 2*B*) revealed a marked adaptation of the tissue to the new function. The coagulum had completely disappeared, and the grafts were covered with a glistening surface indistinguishable from the adjacent normal intima. The grafts were noted to have assumed a thickness approximating that of the aorta proper.

The long-term survivors were kept from seven months to one year; included in this group were five dogs with patches and three with tubes. The specimens (Figs. 1*C* and 2*C*) showed even further favorable changes of adaptation and no evidence of degeneration. Aneurysmal dilatation was not noted in the tube grafts. An aortogram (Fig. 3) was obtained in one animal a year after the insertion of a tube.

Representative specimens have been studied microscopically. In the 5- to 15-day specimens (Fig. 4) the peritoneal layer can be observed to be serving as a substitute for intima. Between this and the rectus sheath is an area of great fibroblastic activity, with thickening of the tissue. The coagulum seen grossly is also demonstrated microscopically.

The intermediate and late specimens (Fig. 5) show an inner surface indistinguishable from normal intima. Beneath this at the site of the earlier fibroblastic activity is an orderly fibrous tissue layer corresponding in thickness to the media of the aorta. The rectus sheath itself remains intact and serves as an accessory supporting layer. The transition from aorta to graft is smooth and almost imperceptible with the hematoxylin and eosin stain. With trichrome stain the graft is easily distinguished from the artery because of the absence of elastic tissue.

SUMMARY AND CONCLUSIONS

In a small series of dogs, defects in the abdominal aorta were repaired with autografts consisting of portions of the posterior rectus sheath with the attached peritoneal surface substituting for intima. The grafts were placed as patches in ovoid defects and as tubes. A high percentage of the patches and a lower percentage of the tubes were successful. Examination of the grafts at various intervals up to one year following implantation has revealed gross and microscopic evidence of adaptation of the tissue for the intended purpose.

It is not suggested that the described method is superior to the use of artery or vein grafts, but it is proposed as a possible substitute in case vessel grafts are not available.

STUDY OF REVASCULARIZATION OF BLOOD VESSEL GRAFTS BY MEANS OF RADIOACTIVE PHOSPHORUS

CLIFFORD L. KIEHN, M.D.
JERROL BENSON, M.D.
DONALD M. GLOVER, M.D.
AND
MARVIN BERG, A.B.
CLEVELAND

CONSIDERABLE evidence has been accumulated by various observers [1] concerning the survival of blood vessel grafts, since the early work of Carrel.[2] It has been shown that both autogenous and homogenous aorta grafts provide satisfactory blood-carrying channels for many months, and perhaps indefinitely. Likewise both homogenous and autogenous grafts of arteries and veins have been used successfully to bridge arterial defects. The percentage of success has been somewhat higher with autogenous than with homogenous grafts. Most observers have reported occasional thrombosis of the grafts regardless of the type employed. Variable factors introduced by methods of storage and preservation of the vascular grafts have been studied extensively.[3]

The mechanism of revascularization of the graft is not entirely certain. It has been shown that in the process of healing part or all of the elements of the graft are infiltrated with granulation tissue, and that the coats of the vessel are gradually replaced by host tissue.[4] The extent to which replacement proceeds and its effect upon function of the graft require further investigation.

The usual criteria of survival of vascular grafts are: (1) effective function of the graft in restoring continuity of the recipient vessel, and (2) histological evi-

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From the Department of Surgery, Plastic Section, University Hospitals and Medical School of Western Reserve University.

1. Deterling, R. A.; Coleman, C. C., Jr., and Parshley, M. S.: Experimental Studies of the Frozen Homologous Aortic Graft, Surgery **29**:419, 1951. Gross, R. E.: Treatment of Certain Aortic Coarctations by Homologous Grafts: Report of 19 Cases, Ann. Surg. **134**:753, 1951. McCune, W. S., and Blades, B.: Viability of Long Blood Vessel Grafts, Ann. Surg. **134**:769, 1951.

2. Carrel, A.: Results of the Transplantation of Blood Vessels, Organs and Limbs, J. A. M. A. **51**:1662, 1908.

3. Peirce, E. C., II; Gross, R. E.; Bill, A. H., Jr., and Merrill, K., Jr.: Tissue-Culture Evaluation of Viability of Blood Vessels Stored by Refrigeration, Ann. Surg. **129**:333, 1949. Macpherson, A. I. S.; Nabatoff, R. A.; Deterling, R. A., Jr., and Blakemore, A. H.: Observations on the Use of Preserved Venous Homografts in Experimental Aortic Defects, A. M. A. Arch. Surg. **63**:152, 1951

4. Swan, H.: Arterial Grafts, editorial, Surg., Gynec. & Obst. **94**:115, 1952.

dences. Both criteria are subject to considerable variability. It has seemed pertinent, therefore, to apply some more objective method to study the survival of vascular grafts. With that intent we have employed the detection of radioactive phosphorus in the present study of arterial grafts.

In previously reported studies upon skeletal grafts we have shown that the presence of P^{32} seems to be a reliable index of vascularity and function of the grafts.[5] Applying a similar method to the present study, we postulated that such a readily diffusible isotope of phosphorus might provide some additional data concerning the vascularization of blood vessel grafts.

PROCEDURE

The method employed was as follows: A section of the carotid artery of the dog was removed, washed in saline solution and immediately replaced in the animal. The vessel on each side of the graft was occluded during the anastomosis by use of double ligatures of No. 00 silk and small segments of rubber tubing. This method is satisfactory if care is exercised in avoiding damage to the arterial wall and intima by constriction. The ligatures should be just tight enough to stop the flow of blood. In several of the anastomoses it was found that a thrombus had formed in the vessel at a point caudad to the graft where the vessel had been ligated.

As these were autogenous grafts, immediately replaced in the animal, it was not necessary to consider the diameter of the two vessels. It is true that the less disparity in size of the vessels the more likely that the anastomosis will remain patent, as well as that eddies, which are conducive to the formation of thrombi, will be prevented. The wound was then closed in layers, and all dead spaces were eliminated as far as possible to prevent the accumulation of serum and extravasated blood which might influence the level of the radioactive isotope in this region.

The animal was then given 300 μc of P^{32} intravenously. All animals in this series were given injections on the day of surgery or at intervals of 10 and 21 days.

The grafts were then removed at 5-, 10-, and 30-day intervals. A section of the graft and control were then assayed for the detection of the isotope. This was done by perfusing the tissue thoroughly to eliminate any adherent material containing the isotope. The specimens were then washed and weighed and the counts per minute per gram were recorded.

Homologous grafts were studied by the same technique. Grafts were removed from the carotid arteries of two dogs and immediately interchanged. The animals were then given 300 μc of P^{32} intravenously, and the grafts and controls were removed at intervals.

Seventeen animals were operated upon and there were 13 survivors.

COMMENT

It seems likely that free blood vessel grafts behave comparably to composites of mesoblastic tissues; mainly, in that certain elements persist for a long time or perhaps permanently, while other elements are gradually replaced by collagenous

5. Kiehn, C. L.; Friedell, H.; Benson, J.; Berg, M., and Glover, D. M.: A Study of the Viability of Autogenous Frozen Bone Grafts by Means of Radioactive Phosphorus, Ann. Surg. **132**:427, 1950.

tissue. The photomicrographs reproduced in Figures 1, 2, 3, 4, 5, 6, 7, and 8, show progressive stages of cellular infiltration from the adventitia and edema of the media at five days, and in subsequent sections ingrowth of fibroblasts and small blood vessels, then, finally, organized connective tissue in the adventitia and part of the media interspersed by many small vascular channels. At the same time it is obvious

Fig. 1.—Photomicrograph of five-day homogenous arterial graft, hematoxylin and eosin stain. P^{32} counts per minute per gram of tissue: control 1,950; graft 1,750. Cellular infiltration is marked in the adventitia and to somewhat less extent in the media. Connective tissue of the adventitia is disintegrating, the muscle fibers of the media are separating, and the intima is largely lacking. (For photomicrograph of control, see Fig. 7.)

that some of the muscle cells of the media persist and probably most of the elastic fibrils. It will be noted in Figure 8, where thrombosis has taken place the vascular reaction and the fibroblastic ingrowth seems to be the most active. As has been

shown by Low-Beer [6] and others, systemic P^{32} tends to concentrate in areas of intense inflammatory or vascular response. The counts recorded in Table 1 tend to confirm this fact. Our observations tend to show that in general the more violent the inflammatory reaction (and the higher the relative count) the poorer the functional result of the vascular graft. The following factors may influence this reaction:

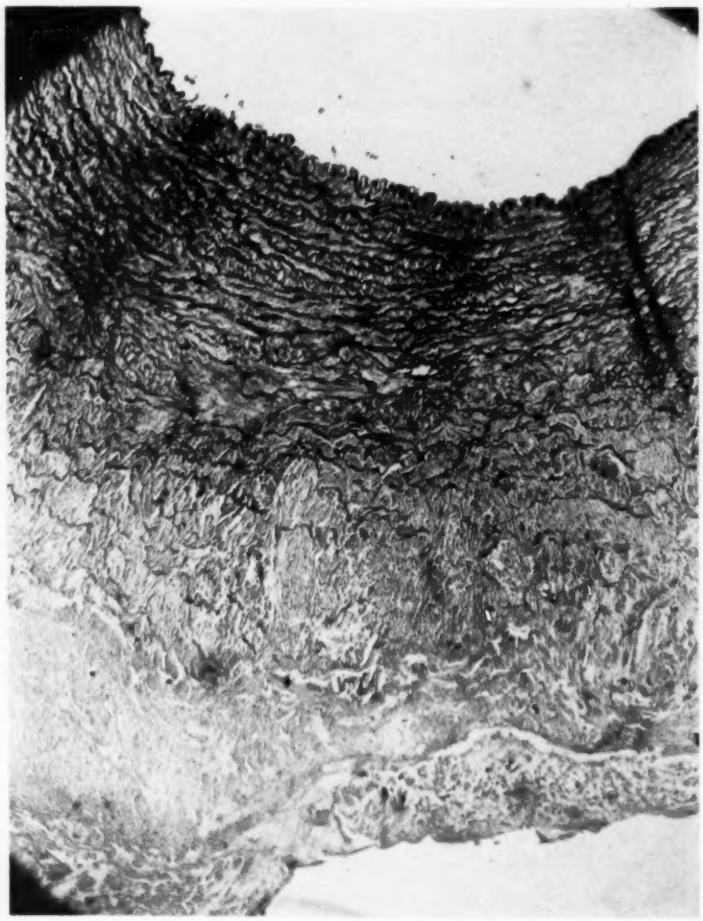

Fig. 2.—Elastic tissue stain of the same section shown in Figure 1. The elastic tissue fibrils clearly shown here have been found to persist throughout the process of replacement by host tissues.

the relative compatibility of the graft, local infection, operative trauma, and cellular changes in the graft prior to placement (for example, those produced by various methods of storage).

6. Low-Beer, B. V. A.: The Clinical Use of Radioactive Isotopes Springfield, Ill., Charles C Thomas, Publisher, 1950.

The detection in the grafts of P^{32} which has been administered intravenously can be accounted for by three methods: (1) by physical adsorption because of the direct contact of the blood containing the P^{32}; (2) by entering the graft through its developing circulation,[7] and (3) by diffusion from the tissue fluids.

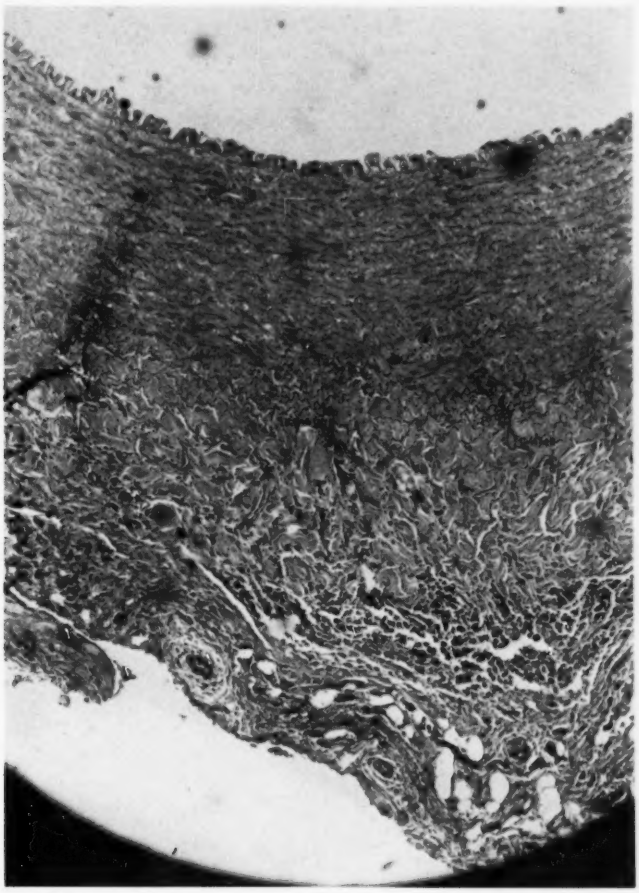

Fig. 3.—Higher-power photomicrograph of 10-day homograft. P^{32} counts per minute per gram of tissue: control 2,960; graft 3,800. Better organized granulation tissue is beginning to invade the adventitia and media, replacing some of the collagenous tissue of the former. A few new blood vessels are appearing.

The data correlated in Table 2 show the trend of isotope counts recorded in Table 1, and perhaps give some basis for interpretation. It is apparent that the average percentage of all the counts on grafts is appreciably higher than that on

7. Conway, H.: Clinical Tests for the Evaluation of Circulation in Tubed Pedicles and Flaps, Ann. Surg. **135**:52, 1952.

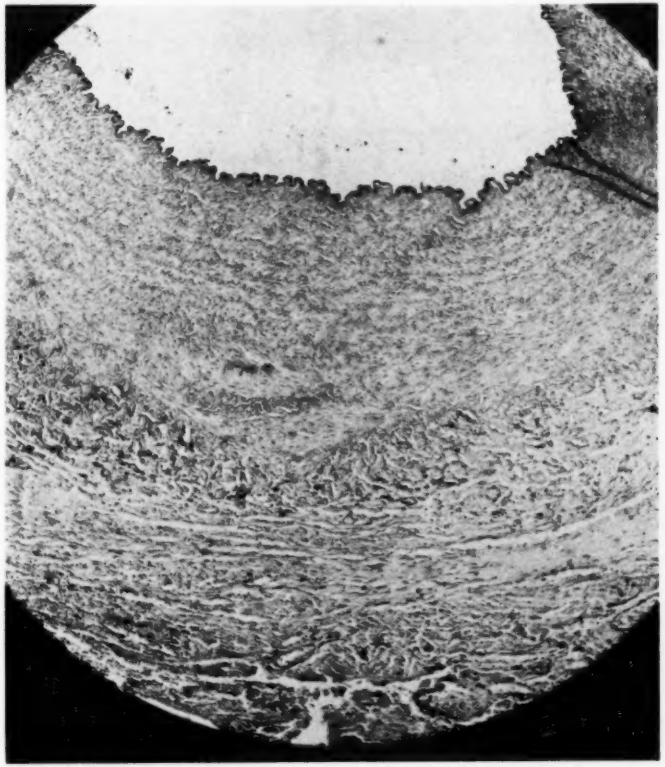

Fig. 4.—Lower-power photomicrograph of 10-day autogenous graft. P^{32} counts per minute per gram of tissue: control 4,270; graft 4,600. This section is quite comparable in most respects to that of the homografts shown in Figure 3.

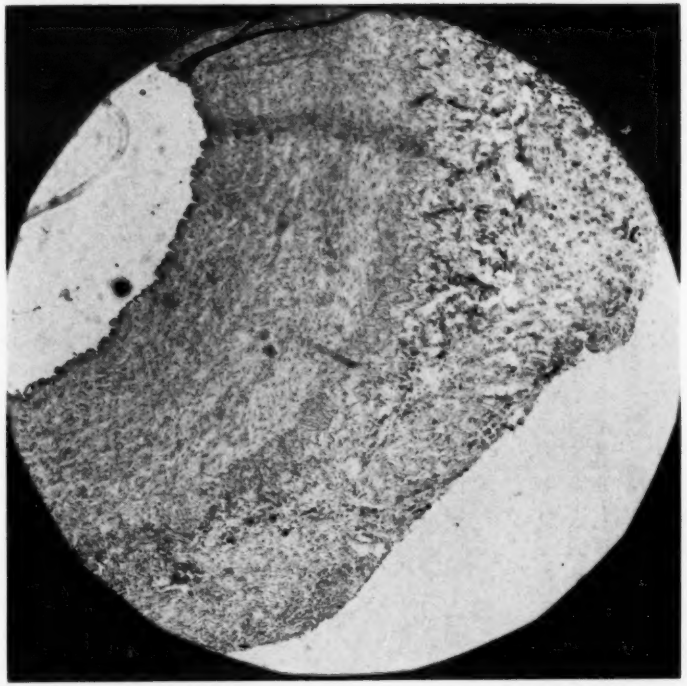

Fig. 5.—Low-power photomicrograph of 30-day homograft. P^{32} counts per minute per gram of tissue: control 1,543; graft, 1,705. Adult collagenous tissue of the host is seen in part of the adventitia while granulation tissue persists in part. Some of the muscle cells of the media have been replaced by connective tissue. The elastic tissue and part of the smooth muscle of the media seem to persist. This was a satisfactorily functioning graft.

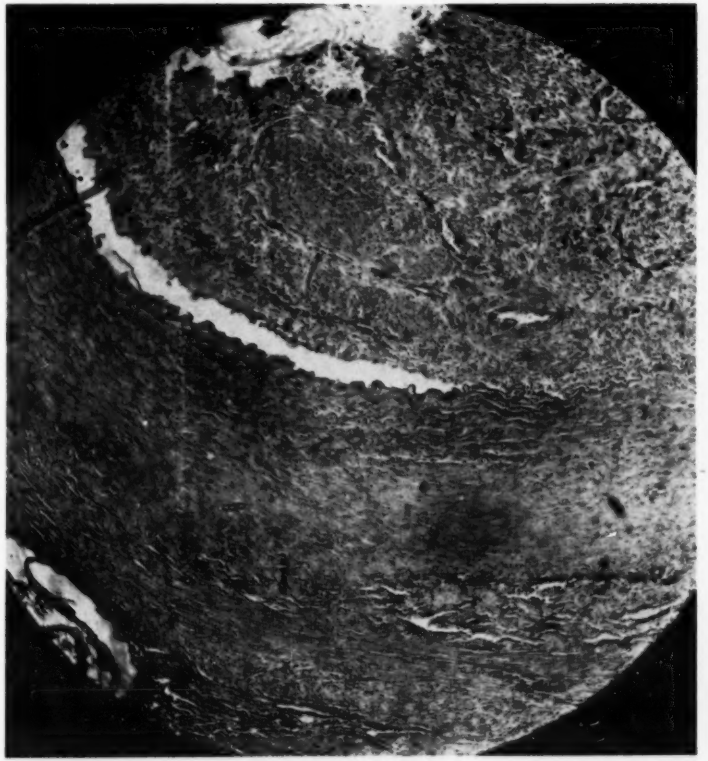

Fig. 6.—Higher power of a 30-day autogenous graft. P^{32} counts: control 202; graft 238. It will be noted that the organization of the adventitia and that of the media are fairly comparable to that seen in Figure 5, but the lumen is filled with an organized and partly recanalized thrombus.

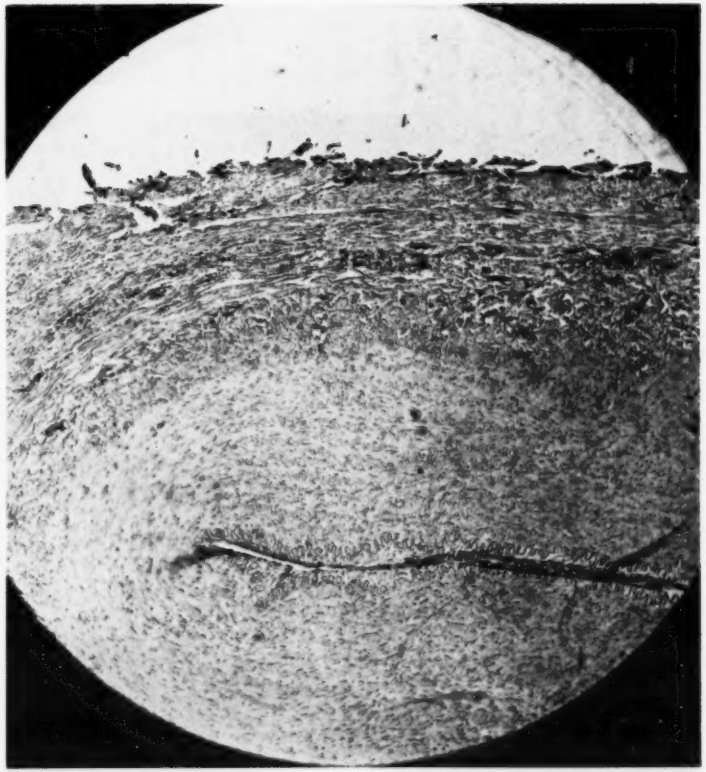

Fig. 7.—Control section of the artery adjacent to the graft shown in Figure 8. All coats of the vessel appear practically normal, with the exception of somewhat greater than usual round-cell infiltration in the peripheral zone of the adventitia. The latter change is probably due to proximity of the operative field.

control arteries (19.4%). The difference between the counts for homografts and those for autogenous grafts is not significant (18.9 and 20.0, respectively, on a percentage basis when compared with controls). Thus from this study we have no evidence favoring autogenous grafts over homografts. This observation may be interpreted as suggesting that tissue of mesoblastic origin excites less violent antagonism upon the part of the host than do tissues of ectodermal origin.

It will be noted also that the average percentages by which the grant counts were higher than the controls was 15.1 for the successful grafts and 35.7 for the grafts which thrombosed. Thus the counts for the satisfactorily functioning grafts

TABLE 1.—*P^{32} In Arterial Grafts*

Type of Graft	Time of Removal, Days	Counts/Minute/Gram	
		Graft	Control
Autogenous	5	1,972	1,952
Autogenous	10	4,600	4,270
Autogenous	30	3,580	1,961
Autogenous	30	1,705	1,665
Autogenous	30	480	465
Autogenous	30	238	202
Homologous	5	2,300	1,940
Homologous	5	1,750	1,950
Homologous	10	3,280	2,135
Homologous	10	3,800	2,960
Homologous	30	302	258
Homologous	30	2,890	2,740
Homologous	30	1,705	1,543

TABLE 2.—*Correlation of Isotope Counts in Arterial Grafts and Controls*

Average Percentages by Which Graft Counts Were Higher Than Controls

All grafts higher than controls by	19.4%
Autogenous grafts by	20.0%
Homogenous grafts by	18.9%
Successful grafts by	15.1%
Thrombosed grafts by	35.7%
Early grafts by	17.8%
Late grafts by	23.1%

averaged lower than the mean of all grafts, while those showing thrombosis, necrosis, interstitial hemorrhage, and inflammatory reaction averaged much higher than the mean. The relatively high uptake of P^{32} in the latter instances is interpreted as being due to the active proliferative response of the small blood vessels to the intense inflammatory reaction, which in turn causes increased diffusion of the isotope into the tissue spaces and cells of the graft. It has been assumed from microscopic studies of Swan [4] and others that the free vascular graft acquires all its blood supply from the host tissues by ingrowth of vessels to the adventitia of the graft. Objective confirmation of this assumption is afforded by the work of McCune, Thistlethwaite, Keshishian, and Blades,[8] who demonstrated by means of

8. McCune, W. S.; Thistlethwaite, J. R.; Keshishian, J. M., and Blades, B.: The Nutrition of Blood Vessel Grafts: An India Ink Injection Study of Their Vascularization, Surg., Gynec. & Obst. **94**:311, 1952.

India ink injections that the only vessels to enter the graft came through the adventitia. By wrapping arterial grafts in cellophane,[8] they were able to show no vascular channels coming in from the lumina of the grafts; but they postulated that the intima and inner layers of the media seemed to be nourished by diffusion from the blood circulating through the graft lumen. The present study seems to confirm, by functional method, the fact that the adventitia is the main source of blood supply,

Fig. 8.—Photomicrograph of section of 30-day autogenous graft. P^{32} counts: control (Fig. 7) 1,961; graft 3,580. Very marked cellular infiltration is seen throughout with beginning disorganization of the media, and a poorly organized thrombus occupies the lumen. Many small blood vessels are seen in the adventitia. The very high isotope count probably reflects the marked inflammatory reaction which is present throughout the graft.

when the isotope detection is correlated with the microscopic picture. Although it is possible that some of the isotope diffuses through the intima, the presumptive evidence is strong that it comes in primarily via the new blood vessels of the adventitia. As further confirmation, it may be noted from Table 2 that the average uptake of the early grafts (5- or 10-day) was significantly less than that of the later (30-day) grafts, the latter having had more time in which to develop good blood

supply from the host. If our premise is correct, one would expect the well vascularized and normally functioning graft to reach, in time, a degree of stabilization at which the uptake of P^{32} would not be significantly different from that of the control.

From the observations recorded it seems likely that the preserved arterial grafts would display a different and perhaps distinctive pattern of isotope diffusion. One might predict that methods of storage which cause tissue disruption or cell destruction would produce in the graft an immediate and marked vascular response which would be reflected by a high uptake of isotope. The method may then be useful in evaluating the various factors which govern the success of the vascular graft.

SUMMARY AND CONCLUSIONS

A method of study by radioactive phosphorus of the vascularization of blood vessel grafts is presented.

It has been shown that factors which increase inflammatory reaction and vascularization also increase the detection of the isotope.

Within the limits of the experiments described, the detection of isotope increases with the age of the graft.

Evidence is presented concerning the character of vascularization of the graft.

The isotopes used were supplied by the Atomic Energy Commission of the School of Medicine of Western Reserve University.

Dr. H. Z. Lund, of the Department of Pathology, assisted in the interpretation of the microscopic sections.

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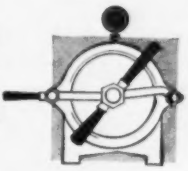

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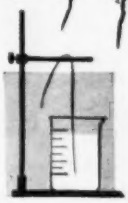

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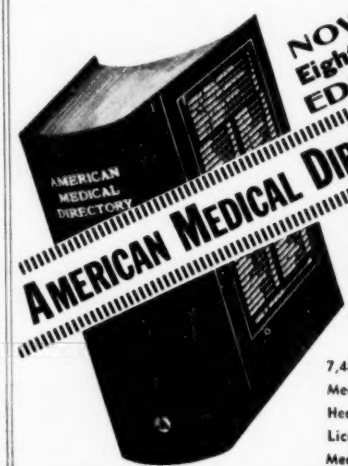

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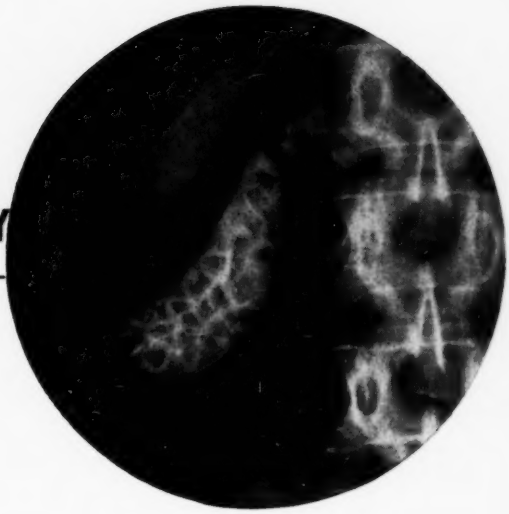

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